THE CANADIAN ANAESTHETISTS' SOCIETY JOURNAL

Vol. 7, No. 1 JAN., 1960

JOURNAL DE LA SOCIÉTÉ CANADIENNE DES ANESTHÉSISTES

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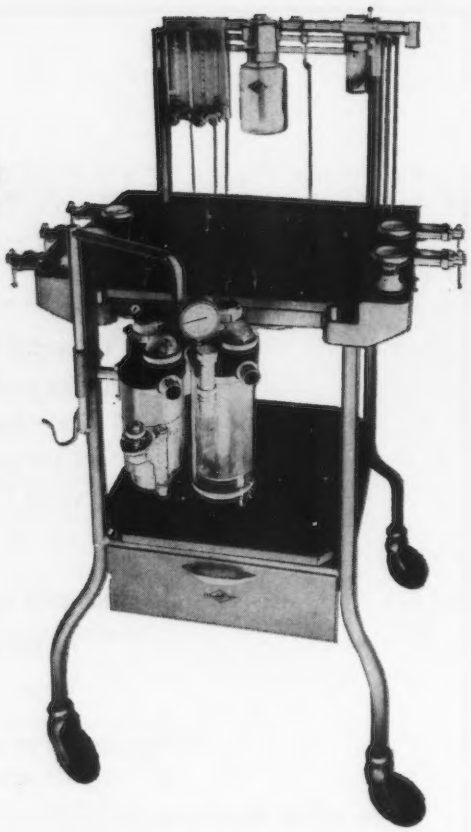

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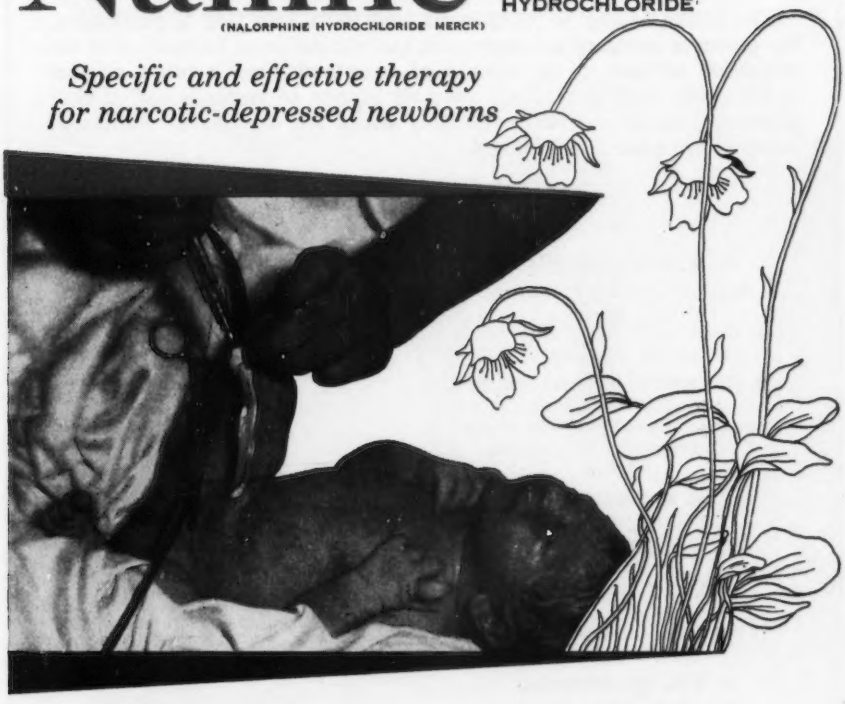

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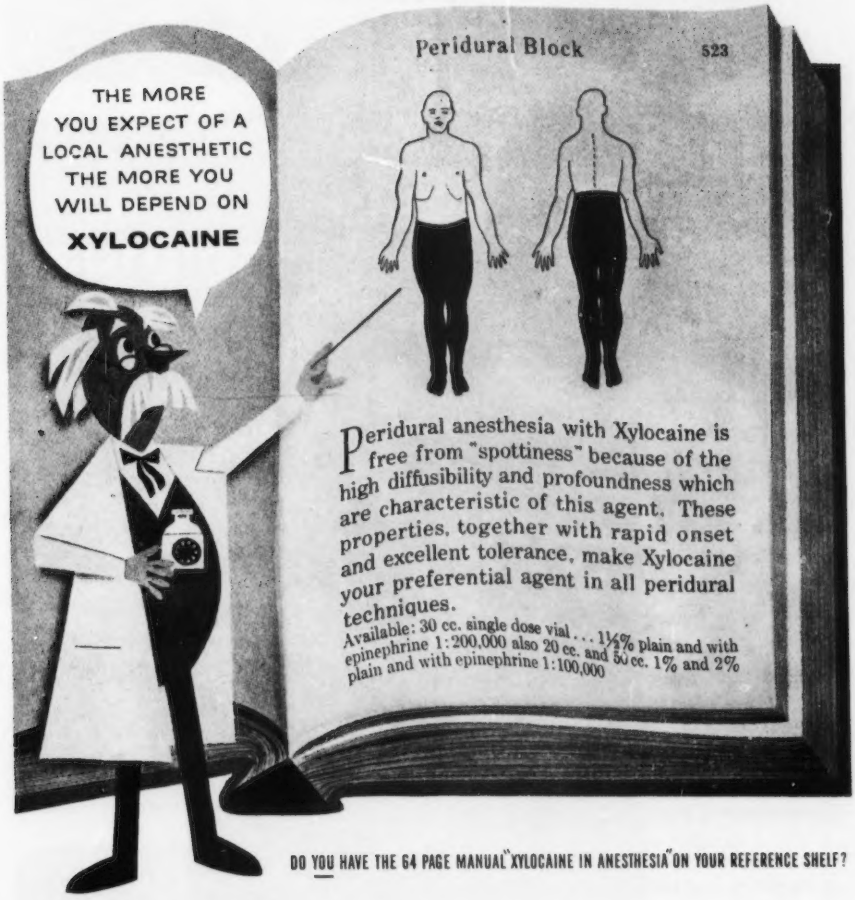
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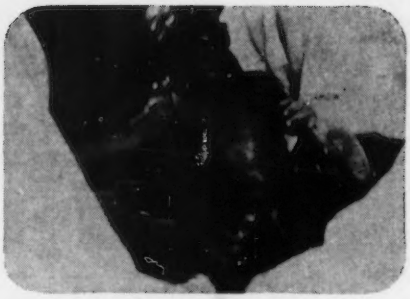

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References: 1. Stone, H.H.: Monographs on Therapy 3:1 (May) 1958. 2. Davies, J.I., and Hansen, J.M.: Clin. Res. Notes 2:5 (May) 1959. 3. Stone, H.H.: Clin. Res. Notes 2:3 (May) 1959.

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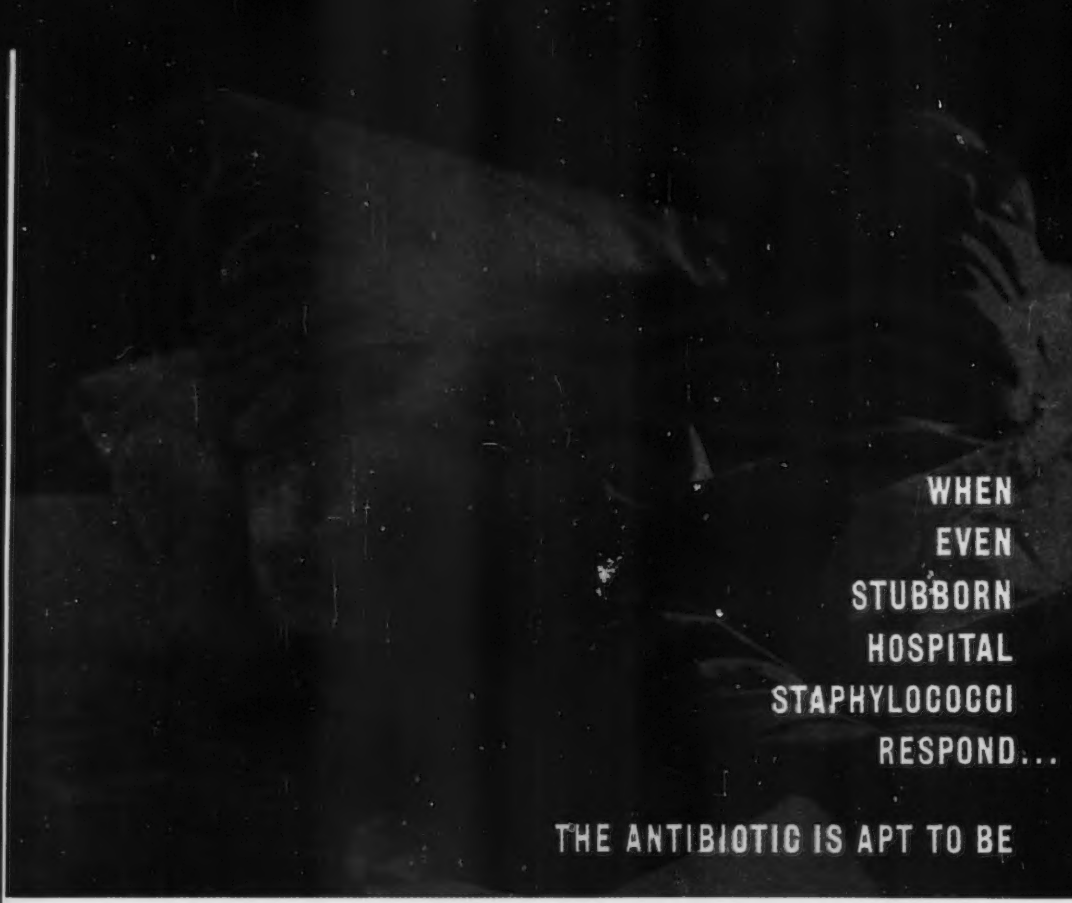

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(1) Goslings, W. R., and Büchli, K.: *Arch. Int. Med.* 102:691, 1958. (2) Flippin, H. F.: *Am. Pract. & Digest Treat.* 10:39, 1959. (3) Borchart, K. A.: *Antibiotics and Chemother.* 8:564, 1958. (4) Fischer, H. G.: *Deutsche med. Wchnschr.* 84:257, 1959. (5) Holloway, W. J., & Scott, E. G.: *Delaware M. J.* 30:175, 1958. (6) Suter, L. S., & Ulrich, E. W.: *Antibiotics & Chemother.* 9:38, 1959. (7) Markham, N. P., & Shott, H. C.: *New Zealand M. J.* 57:55, 1958. (8) Ditmore, D. C., & Lind, H. E.: *Am. J. Gastroenterol.* 28:378, 1957. (9) Schneierson, S. S.: *J. Mt. Sinai Hosp. New York* 25:52, 1958. (10) Godfrey, M. E., & Smith, I. M.: *J.A.M.A.* 166:1197, 1958.

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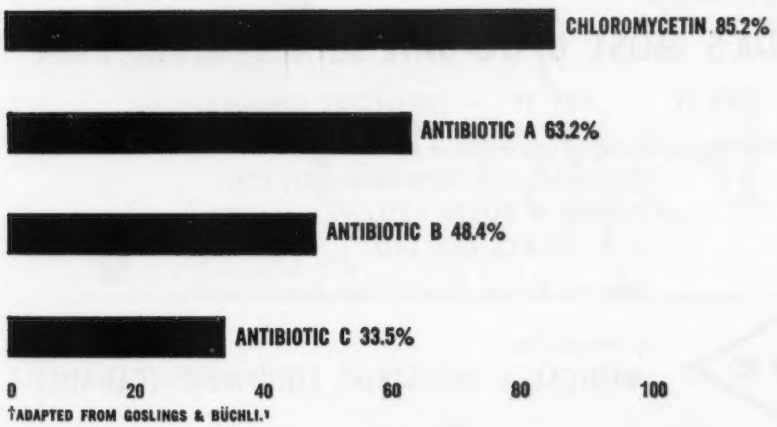

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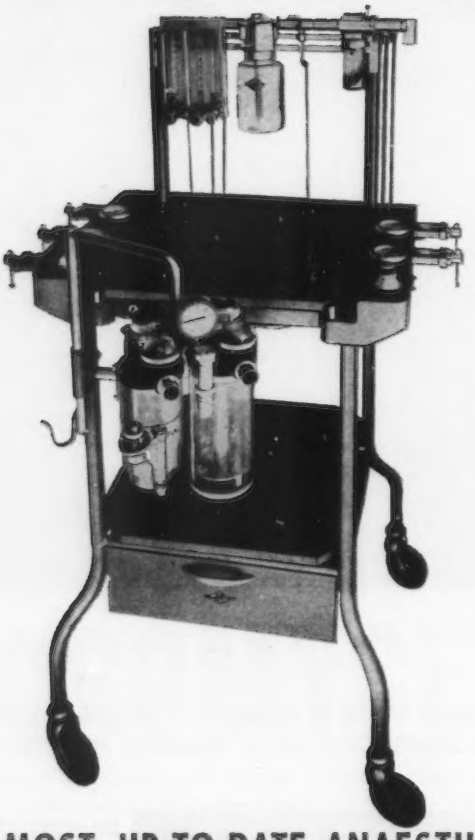

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1. Webster, C.W., C.M.A.J. Oct. 1953.

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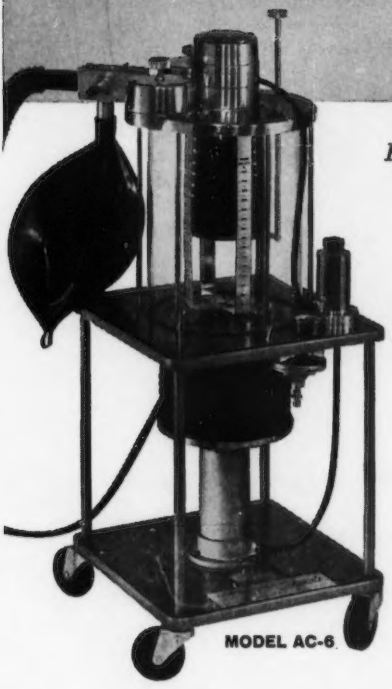

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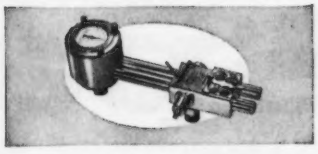

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A WELCOME

ALL THOSE INTERESTED in the World Federation of Societies of Anaesthesiologists will join with us in welcoming the American Society of Anesthesiologists into the Federation. The stated objective of the Federation "to provide better anaesthesia for all the peoples of the world" was prejudiced so long as the largest and most influential of the national societies of anaesthesiologists held aloof from the world organization. Individual members of the American Society of Anesthesiologists have done a great deal in the past to improve the standards of anaesthesia throughout the world, and these, too, must be happy that the A.S.S. has officially accepted responsibilities in the international sphere.

BIENVENUE

Toutes les personnes intéressées dans la Fédération Mondiale des sociétés d'anesthésiologistes voudront se joindre à nous pour souhaiter la bienvenue dans la Fédération à la Société Américaine des Anesthésiologistes. L'objectif connu de la Fédération "procurer une meilleure anesthésie à tous les peuples de la terre" subissait un préjudice du fait que la plus considérable et la plus influente des sociétés nationales des anesthésiologistes était tenue à l'écart de l'organisation mondiale. Certains membres de la Société Américaine des Anesthésiologistes, dans le passé, ont beaucoup fait pour améliorer les standards de l'anesthésie dans le monde; eux aussi, aujourd'hui, doivent être heureux que la Société Américaine des Anesthésiologistes ait accepté officiellement des responsabilités dans le domaine international.

POSTURE AND ANAESTHESIA[1]

DAVID M. LITTLE, JR., M.D.[2]

. . . I directed that chloroform be administered to him for the purpose of performing an operation upon the rectum. . . . the patient soon became excited by the chloroform, as is usually the case, but not beyond a degree that I have often observed. Shortly after, he became more tranquil. . . . [he] was placed upon his side, and the operation performed, which consisted in the removal of two external tumours, and the tying of one internal tumour.

At this moment my attention was arrested by my assistant's calling for a wet cloth: on examining the patient, I found his face and neck of a livid hue or colour, the eyes turned upward, the pulse imperceptible at the wrist, and the whole body relaxed; after two or three gasps, he ceased to breathe. Every means was promptly used to restore the deceased, but without effect.

THESE WERE the words of Gurdon Buck, Jr., attending surgeon to the New York Hospital, describing a death that occurred on January 19, 1849 (1). John Griffith, aged 31, a seaman in the American Navy, was having some haemorrhoids removed when the catastrophe occurred. This was only one of a number of chloroform deaths that occurred soon after the introduction of that agent into clinical anaesthesia practice, but it was perhaps the first such death in which posture played an important aetiological role. It is naturally difficult to reconstruct the details of an event that took place over a century ago, but it was undoubtedly more than mere coincidence that an anaesthesia which had been progressing in normal—if somewhat hectic—fashion, suddenly deteriorated when the patient was moved into the lateral position. It may be surmised that placing the patient on his side led to the pooling of blood in peripheral vessels, which were dilated because of direct depression of the vasoconstrictor centre(s) of the medulla. There no doubt followed a decrease in venous return, a fall in cardiac output, a fall in mean aortic pressure, and therefore an insufficient coronary flow to maintain myocardial oxygenation. These circulatory effects may well have been abetted by the effects of the lateral position upon respiration, decreasing ventilation and thus leading to further reduction of oxygenation and the development of hypercarbia. The combination of suboxygenation of the myocardium, the existence of a state of hypercarbia, and the presence of a halogenated hydrocarbon, which is toxic to the heart, is quite sufficient explanation for the tragedy that ensued. All this is, of course, pure conjecture, and the precise extent to which turning the patient on his side influenced the disastrous course of events remains unknown; but in retrospect it seems obvious that it had considerable effect, and that just possibly it was the decisive factor. Today, one hundred years later, posture during anaesthesia continues to be an important agent in anaesthetic morbidity and mortality.

[1]Presented before the Sectional Meeting of the American College of Surgeons, Montreal, P.Q., April 6, 1959.

[2]From the Department of Anesthesiology, Hartford Hospital, Hartford, Conn.

The position of the patient during operation is almost always prescribed by the type of surgical intervention being performed. It is usually regarded in a most offhand way by the surgeon and anaesthesiologist alike; and thus not infrequently leads to thoroughly undesirable effects, both during and after surgery. There are a number of reasons why this is so. In the first place, some of the positions devised for the facilitation of surgical procedures simply defy the muscular and skeletal mechanics possible to the human body. In the second place, all postures—to a greater or lesser extent—affect such vital functions as circulation and respiration—which depend, respectively, upon the movement of blood and air. Certain postures affect these functions in a most adverse manner, indeed. And, finally, the position of the patient during anaesthesia may lead to injurious effects or actual injuries which the patient would defend himself against if he were not under anaesthesia, but possessed of his normal sensations and compensatory reflexes. Nicholson has given this latter point much thoughtful consideration, and has emphasized that the effects of position upon the patient during surgery deserve the cognizance of the entire surgical team, and particularly of the anaesthesiologist and surgeon, who are obligated to protect the welfare of their patient (2).

Respiration

The effects of posture on respiration may include changes in pulmonary blood volume (3), in the compliance of the lungs (4), and in the intrapulmonary distribution of the inspired air (5); but the most important effects are due to limitations in the expansion of the lungs from simple mechanical interference with their normal movements (6). During normal respiration in the unanaesthetized individual, the thoracic cage enlarges in all directions except posteriorly: it expands vertically by the downward descent of the diaphragm, transversely by a "bucket-handle" upward and lateral movement of the ribs, and anteroposteriorly by a similar upward but forward movement of the ribs and sternum. The effect of various surgical positions in limiting these movements has been studied in normal conscious subjects by measuring the vital capacity with a standard Benedict-Roth spirometer (7, 8, 9, 10) (Fig. 1). Most studies of this type have shown that the highest readings are obtained in the sitting position—since this posture does not interfere with either the movement of the diaphragm, or the motions of the ribs and sternum. All other surgical positions produced some decrease in the vital capacity (11). The supine position produced a 9.5 per cent decrease in the vital capacity, owing to the pressure of the abdominal viscera against the diaphragm, restricting its downward motion. The reverse Trendelenburg position exhibited this same effect, but to a lesser degree, the decrease in vital capacity being 9.0 per cent. The prone position caused a 10 per cent decrease in vital capacity, mainly owing to a restriction in the anteroposterior expansion of the lungs, but also owing to some impairment in the movement of the anterior abdominal wall and hence the diaphragm, as well as in the transverse expansion of the lungs—since restriction of the motion of the ribs in an anterior direction will also inevitably restrict their motion in a lateral direction to some extent, and vice versa. The lateral position also caused a 10 per cent decrease in vital capacity,

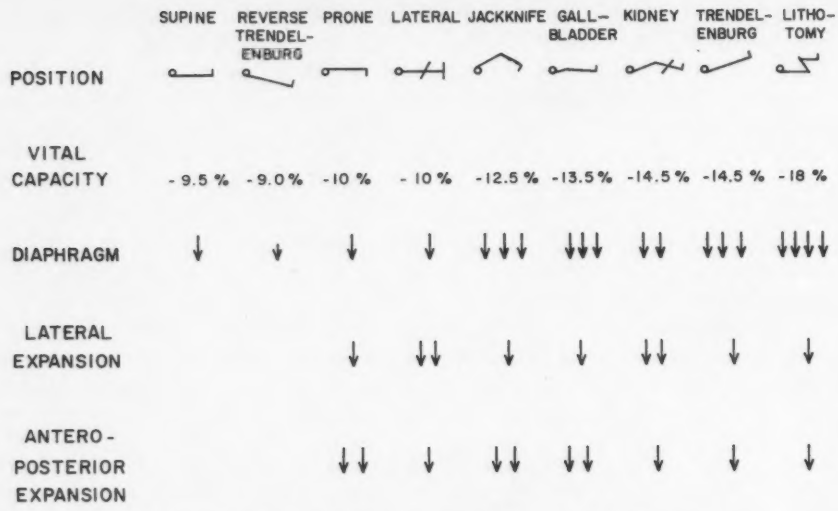

FIGURE 1. THE RESPIRATORY EFFECTS OF SURGICAL POSTURES

The effect of position in the normal, conscious subject as it effects vital capacity, diaphragmatic movement, lateral expansion of the chest, and anteroposterior expansion of the chest.

mainly because of a restriction of lateral expansion of the side of the chest on which the subject was lying, but also because of some impairment of the movement of the diaphragm on that side, as well as a concurrent impairment of the anterior rib motion that accompanied restriction of lateral rib motion on that side. The jack-knife position produced a 12.5 per cent decrease in vital capacity, owing mainly to restriction of diaphragmatic movement, a lesser but significant impairment of anteroposterior expansion, and some concurrent impairment of lateral expansion. Raising the gallbladder rest in the supine subject decreased vital capacity 13.5 per cent, again mainly because of impairment of diaphragmatic movement and anteroposterior expansion, with some concurrent restriction of lateral expansion. The kidney position produced a 14.5 per cent decrease in vital capacity, owing to impairment of thoracic cage enlargement in all directions, although anteroposterior expansion is affected less perhaps than the other two movements of the lungs. The Trendelenburg position also led to a 14.5 per cent decrease in vital capacity, owing mainly to impairment of diaphragmatic movement and a restriction of the volumetric expansion of the lungs because of increased pulmonary blood volume. Lithotomy position produced the greatest decrease in vital capacity, 18 per cent, as a result of marked restriction of diaphragmatic movement and a great increase in pulmonary blood volume.

It must be realized that measurements of alterations in vital capacity, while indicating the degree of restriction of lung expansion to some extent, do not necessarily reflect the condition of respiration during anaesthesia since vital capacity—by definition—measures the maximum inspiratory effort in the conscious,

co-operating individual. Tidal volume has, therefore, been employed as a better measure of the respiratory effects of the various surgical postures in the anaesthetized patient (12, 13). The results of this type of measurement reveal a number of minor differences in comparison to the studies of vital capacity in the conscious subject, but the general trend is very similar. Both a 10° Trendelenburg position and the lithotomy position caused a 3 per cent decrease in the tidal volume of the anaesthetized subject; the lateral position caused an 8 per cent decrease; prone position with support an 11 per cent decrease; 20° or 30° Trendelenburg a 12 per cent decrease; the prone position without support, the kidney position, the gallbladder position with a 10° head-up tilt, and the lithotomy position with 10° Trendelenburg, 14 per cent decreases; lithotomy position with 20° Trendelenburg a 15 per cent decrease; and the gallbladder position with the gallbladder rest raised a 24 per cent decrease (12).

It is worth pointing out that the adverse effects of these restrictions of respiratory movement and ventilation are not often immediately apparent from the clinical viewpoint, but rather build up slowly during the course of operation. The gradual onset of fatigue of the respiratory muscles in overcoming the restrictions to chest movement, for instance, is obviously correlated with the duration of time that those muscles must work against such embarrassment. Much less obvious, but of possibly greater importance, is the effect of the hypoventilation produced by the impairment of respiratory movements. If the tidal volume is 500 cc. in the normal conscious subject, this will be reduced to about 400 cc. during anaesthesia. Only 200 cc. of this amount is effective for ventilation, since 150 cc. represents anatomical dead space and a further 50 cc. represents mechanical dead space in the anaesthesia mask. When the tidal exchange is reduced 25 per cent or 100 cc., by raising the gallbladder rest, alveolar exchange will be reduced to only 100 cc. (12). This is, of course, insufficient to maintain normal ventilation, and in time will lead to hypoxia and hypercarbia. Therefore, the longer that an adverse surgical posture is maintained during operation, the greater will be the deleterious effects of respiratory fatigue and hypoventilation.

Circulation

The effects of the various surgical postures upon the circulation, on the other hand, are much more apt to be immediately apparent and dramatic in onset (Fig. 2). The individual postures possess their own individual hazards, but they all share the circulatory danger of movement of the anaesthetized patient, because of the vasodilatation and obtundation of compensatory cardiovascular reflexes which are manifest during anaesthesia (14, 15). Thus, a number of these postures may be tolerated by many normal individuals, but movement into, or out of, those postures during anaesthesia—and particularly during deep anaesthesia—can lead to a failure of the compensatory mechanisms to effect the necessary redistribution of blood volume, a reduction of venous return, a decrease in cardiac output, and cardiovascular collapse (16). This sequence of events can be seen when the patient is moved from the supine to the lateral position or from the lateral to the supine position, when the head and feet are lowered in the lateral

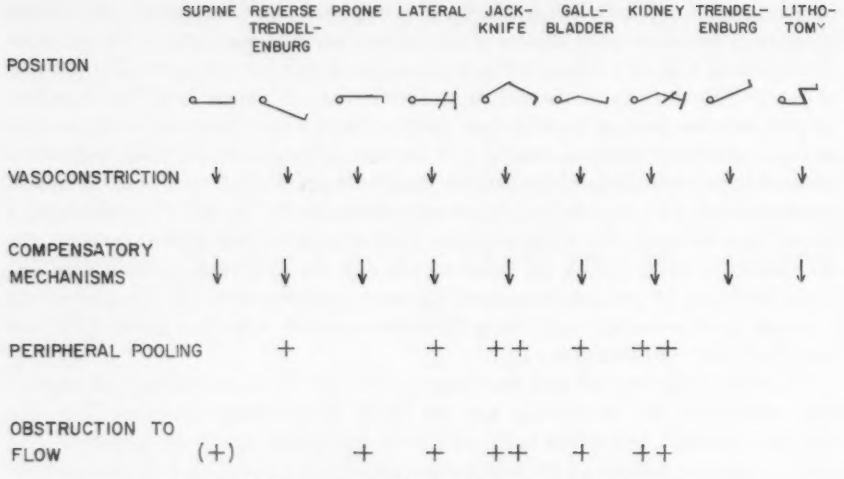

FIGURE 2. CIRCULATORY EFFECTS OF SURGICAL POSTURES

The effect of position in the anaesthetized subject on such circulatory factors as vasoconstriction, compensatory mechanisms, peripheral pooling, and obstruction to flow.

position and so forth (17). The lower the cardiovascular reserve—whether on the basis of heart disease, decreased blood volume, or peripheral vascular decompensation—the more intense the adverse response to change of position of the anaesthetized patient; but even the healthiest individual may be jeopardized by a change in posture under anaesthesia since the homeostatic compensatory mechanisms are depressed by anaesthetic drugs, and such active vasopressor reflexes as the carotid and aortic mechanisms are no longer fully effective (18).

In addition to this common danger to the circulation inherent in the movement of the anaesthetized patient, however, there are certain specific circulatory effects peculiar to individual postures. The supine position, for instance, is perhaps the most benign to the cardiovascular integrity during anaesthesia, yet even this posture may possess the danger of marked venous obstruction if a large intra-abdominal tumour mass is present, as in the pregnant woman: normotension may be present when the patient is in the lateral position, but then rather profound hypotension may occur when the patient is placed in the supine position, owing to obstruction of flow in the inferior vena cava by the gravid uterus (19). The reversed Trendelenburg position, under the conditions of vasodilatation which pertain during anaesthesia, permits considerable peripheral pooling in the lower extremities, which can amount to several hundred cubic centimetres or more of blood (14). The prone position is also capable of disturbing cardiovascular dynamics, particularly in the obese individual in whom the abdominal contents and a large panniculus provide a firm mass to obstruct caval flow. The lateral position permits peripheral pooling in the dependent half of the body, and also some obstruction to flow, particularly if the patient is lying on the right side. The jack-knife position is one of the most pernicious surgical postures from the circulatory

point of view since there is marked peripheral pooling in the two dependent portions of the body, and quite severe obstruction to flow in the inferior vena cava. The gallbladder position with the gallbladder rest raised permits some degree of peripheral pooling and, depending upon the exact position of the gallbladder rest, can also cause quite marked obstruction to caval flow. The kidney position produces both marked peripheral pooling and marked obstruction to flow; the latter is particularly apparent when the patient is on the right side, but it may also occur when the patient is on the left side; and, in addition, in this instance, there may be actual interference with heart action due to pressure on the lower chest and shift in the position of the heart. The Trendelenburg position, particularly if it is a steep Trendelenburg position, certainly produces pooling in the upper half of the body, which is traditionally thought of as salutary in terms of circulation to the vital organs; however, it is not always so, for undue engorgement of the vessels in the thorax and mediastinum, combined with the pressure of the viscera against the diaphragm, may actually decrease stroke volume and cardiac output. Furthermore, it is not necessarily true that a steep Trendelenburg position will safeguard the integrity of the circulation through the brain, as the venous and cerebrospinal fluid pressures within the rigid confines of the cranial vault may actually interfere with the inflow of arterial blood and lead to a stagnant type of hypoxia. The lithotomy position also shifts a large quantity of blood from the lower part of the body, and can produce obstruction to flow owing to the pressure of the abdominal viscera against the great vessels and the diaphragm.

It should be emphasized again that in the normal healthy individual the greatest danger of these abnormal surgical postures is during and after movement into or out of a posture. The lithotomy position is a most pertinent example. When the legs are lowered from the lithotomy position, 500, 600, or up to even 800 cc. of blood may be drained into the lower extremities, depending upon the depth of anaesthesia and the degree of vasodilatation. Depression of the compensatory cardiovascular reflexes, such as the carotid and aortic mechanisms, under such circumstances will lead to temporary cardiovascular collapse with rather severe hypotension. In the normal, healthy individual, this collapse may be momentary and even unobserved; but in the poor risk individual, with low cardiovascular reserve, such collapse may be severe and dangerous. Further, in these poor risk individuals, even less gross insults than the example cited can lead to deterioration of circulatory efficiency. The obese individual with low cardiovascular reserve, for instance, when placed in the jack-knife position, may gradually suffer a circulatory deterioration over the course of the first few minutes of assuming that posture, and not regain cardiovascular status quo until removed to a more normal posture.

Skeletal and Nervous Systems

The unpleasant effects of surgical postures upon both respiration and circulation are most apt to be exhibited during or very soon after operation. This is not always true, of course, for certainly postoperative atelectasis may be the direct result of restriction of chest movement during operation, but not become manifest until well into the postoperative period. The effects of malposition upon the

skeletal, muscular, and peripheral nervous systems, on the other hand, are most apt to become apparent in the postoperative period and during convalescence; they are often masked by postoperative pain and illness, and therefore are often overlooked by the physician (2). These injuries are due to stresses, strains, and stretches, and are most likely to occur when the surgical posture is extreme, when muscular relaxation is profound, and when the duration of operation is long (20). The most common example of this type of sequelae is the backache often complained of following, and attributed to, spinal anaesthesia: such backache is due to the fact that spinal anaesthesia produces such complete relaxation that the legs lie perfectly flat upon the operating table, and the stretch upon the ligaments and muscles of the lower spine is sufficient to produce postoperative backache.

Injuries to peripheral nerves are also the result of stresses and strains, particularly pressure, and have received such widespread publicity that they need only be mentioned briefly at this time (21, 22). The brachial plexus is susceptible to stretch when the arm is hyperextended, to pressure from a misplaced shoulder brace, or to stretch encountered in the old method of suspending the patient with wrist straps in steep Trendelenburg. The radial, ulnar, and median nerves are also susceptible to pressure injuries as they traverse the brachium, and the classic example of this type of complication is seen following the use of the prone position in which the patient's arm overhangs the metal edge of the operating table. In similar fashion, the peroneal nerve may be injured by pressure in the lithotomy position when it is compressed between the metal standard holding the leg stirrups and the head of the fibula. Recovery from such nerve injuries is often complete if there has been no actual disruption of the anatomic and functional continuity of the nerve fibres; however, even when recovery is complete, it often takes a long time.

The Prevention of Untoward Effects

Now it must be emphasized that, whatever the undesirable effects of some of the more extreme postures employed during operation, the postures themselves are necessary and desirable for the facilitation of the surgery or they would have been abandoned long since. From the surgeon's point of view, the role of a particular position in terms of time required to complete the surgery, visibility for the definitive procedure, and ease of manipulation of tissues and structures is often all-important. These surgical postures, in short, however deplorable they seem in terms of their effects upon respiration, circulation, and the skeletal and nervous systems, cannot be wished away; their disadvantages, therefore, must be ameliorated by active countermeasures. A great deal more can be accomplished in this regard than is generally realized.

The respiratory depression inherent in many of the surgical postures, for example, can in many instances be alleviated by the combined use of an endotracheal tube and assisted or controlled respirations (12, 13). Endotracheal intubation is desirable during the use of assisted or controlled respirations to prevent inflation of the stomach, but it also serves to offset the respiratory effects of the surgical position to a certain extent by reducing the dead space (both anatomical and mechanical) by some 75 cc. and thus contributes this amount to effective

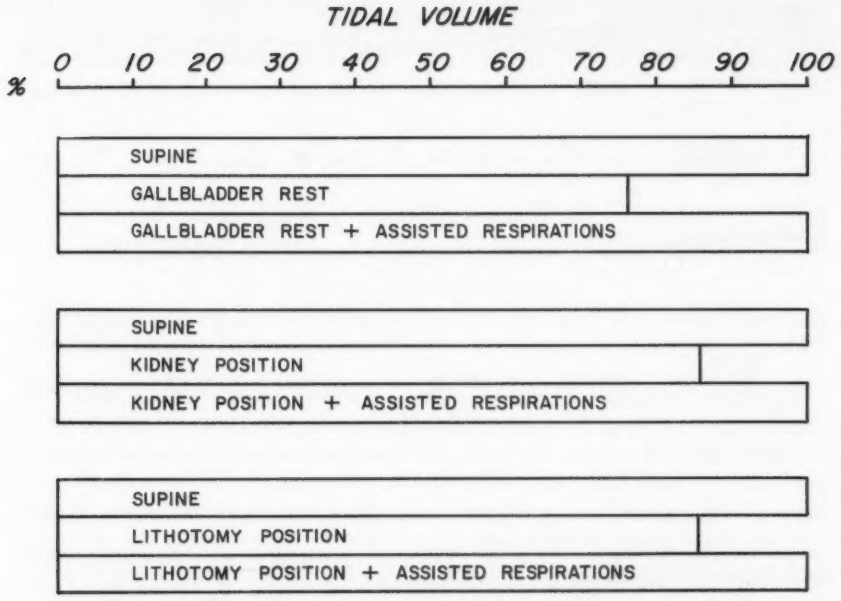

FIGURE 3. EFFECT OF ARTIFICIAL VENTILATION IN CERTAIN SURGICAL POSTURES

The upper set of bar graphs depicts the reduction in tidal volume when the gallbladder rest is raised, and the return to control value of tidal volume when respirations are assisted; the *middle* set of bar graphs depicts similar data for the kidney position; and the *lower* set of bar graphs depicts the same data for the lithotomy position.

alveolar ventilation. Artificial ventilation, by assisted or controlled respirations, can in many instances totally offset the respiratory depressant effects of surgical positions (Fig. 3). The use of the gallbladder position and rest, for instance, was found to reduce respiratory volume by 24 per cent in comparison with the supine position; with the use of assisted respirations, however, respiratory volume could be maintained at a perfectly normal level (12). The same is true of the use of the kidney rest in the lateral position, which produces a 14 per cent reduction of tidal volume; the tidal volume can be restored to, and maintained at, a normal level by the use of assisted respirations (12). A final example is the lithotomy position, which also produces a 14 per cent reduction in respiratory volume, a decrease that can be entirely offset by the use of assisted or controlled respirations (12). It is evident, therefore, that in the vast majority of instances the untoward effects of surgical posture upon the respiratory system can be in large measure offset by the very simple expedient of assisting or controlling respirations to restore respiratory volume to normal.

The most dangerous of the circulatory effects associated with the various surgical postures is the possibility of cardiovascular collapse at the time of positioning the patient on the operating table at the beginning or end of operation. Much of the danger associated with these manoeuvres can be overcome by the simple expedient of moving the patient slowly, gently, and smoothly. Movement

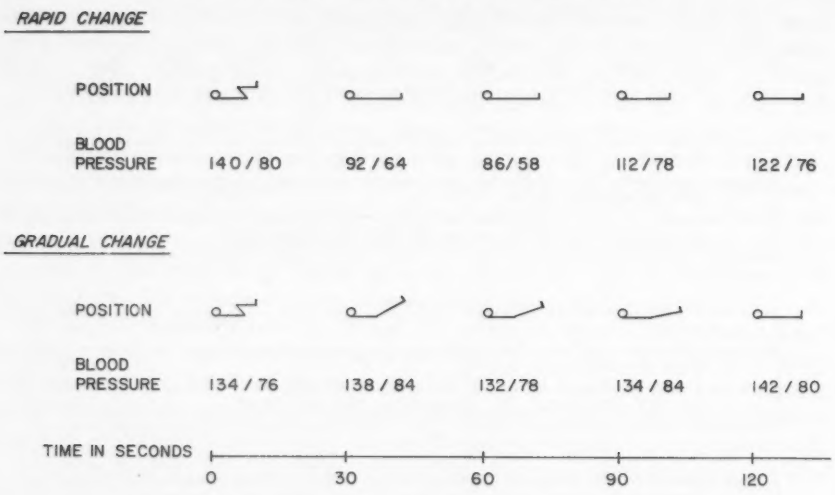

FIGURE 4. CHANGING THE POSITION OF THE ANAESTHETIZED PATIENT

When the anaesthetized patient is moved from the lithotomy to the supine position rather rapidly, there is a precipitous fall in blood pressure that persists for as long as 2 or more minutes. When the same patient, maintained at the same depth of general anaesthesia, is moved from the lithotomy to the supine position very gradually, there is no detectable change in blood pressure.

of the patient from the lithotomy position, for instance, if done in a straightforward manner and accomplished in rapid fashion, will usually lead to some lowering of blood pressure (Fig. 4). In some instances, this fall will be so small and so momentary that it will be detected only with a continuous intra-arterial tracing. In other instances, as seen in Figure 4, a measurable drop in pressure will occur and persist for a minute or more. In a few patients, in whom the cardiovascular reserve is low, the fall in blood pressure will be sufficiently severe and sufficiently long in duration to produce real collapse. On the other hand, if the position is changed very slowly over the course of a minute or more the shift from the lithotomy to the supine position can be accomplished with almost no change in blood pressure. When one is dealing with very poor risk patients such as some of the candidates for mitral commissurotomy, the patient can be positioned on his side initially, following which the induction of anaesthesia and the accomplishment of endotracheal intubation can be carried out with the patient in the lateral position. This technique, while more difficult technically, will serve to prevent some of the unfortunate circulatory changes associated with the movement of the patient from the supine to the lateral position during a depth of anaesthesia that has been sufficient to permit endotracheal intubation.

The specific countermeasures which may be taken to avoid or offset the respiratory and circulatory effects of surgical postures are valuable, but it must be emphasized that the most important step which can be taken in regard to posture during anaesthesia and operation is the proper use of the position demanded by the specific operative procedure. There is a right way and a wrong way to accom-

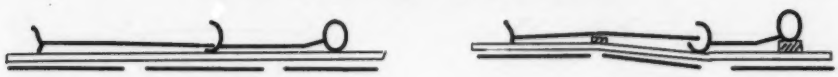

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FIGURE 5. THE SUPINE POSITION

The supine posture should be modified to provide some flexion of the knees, some flexion of the thighs, and some flexion of the neck.

plish each specific surgical posture, as indicated in the following examples.

The supine position, for instance, should consist of more than just unceremoniously removing the patient from the stretcher onto a flat operating table (Fig. 5). The posture should be modified to provide some flexion of the knees, some flexion of the thighs, and some flexion of the neck. These are easily accomplished by the rather simple expedients of placing a small bolster under the popliteal spaces, appropriate breaking of the operating table, and raising the head onto a small pillow.

The prone position should also be something more than an unceremonious rolling of the patient from the stretcher to a face-down position on the operating table (Fig. 6). Instead of the patient lying flat, a thick pad or pillow should be placed under the iliac crests, so that the body is suspended from the crests; a blanket roll should be put under each shoulder girdle, to give free motion to the anterior chest and the diaphragm; and a small bolster should be used under the feet, to allow slight flexion at the knees and hips.

The gallbladder position—in this, the Age of Muscle Relaxants—is being used a great deal less than previously; but the gallbladder position with a high gallbladder rest need not be used at all (Fig. 7). Resort to a modified supine position, with a small pad or pillow under the bed of the gallbladder on the right side of the patient, should suffice to satisfy the most discriminating surgical taste for exposure.

INCORRECT CORRECT

FIGURE 6. THE PRONE POSITION

A thick pad or pillow should be placed under each iliac crest, so that the body is suspended from the crests; a blanket roll should be placed under each shoulder girdle; and a small bolster should be used under the feet.

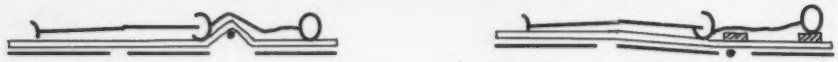

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FIGURE 7. THE GALLBLADDER POSITION

The gallbladder rest should not be used at all. Use the modified supine position with a small pad or pillow under the right side of the patient, directly below what will be the site of the gallbladder bed.

The kidney position can also be modified in such a way that surgical exposure is adequate without placing the patient in an extreme posture (Fig. 8). The patient is positioned with the kidney-lift supporting the crest of the ilium; a large pillow is placed between the patient's knees to prevent pressure, the lower leg being flexed at both the hip and the knee; and the operating table is broken immediately under the flank.

The Trendelenburg position, as originally described, should always be modified (Fig. 9). Most important, perhaps, is the avoidance of shoulder braces or wrist straps, employed to prevent the patient from slipping; instead, the mattress should be taped to the operating table—which will effectively prevent both the mattress and the patient from slipping. Of almost equal importance is the avoidance of 30° Trendelenburg when 20° will suffice, and the avoidance of 20° Trendelenburg when 10° will suffice! The table should be broken to provide flexion at both the hips and the knees, and a small pillow should be placed under the head to allow flexion of the neck.

The lithotomy position should also be something more than just a patient with legs up in stirrups (Fig. 10). Great care must be taken, of course, to pad pressure spots on the feet or lower legs. In addition, the use of bolsters or pillows at the side of, and slightly under, the hips will aid flexion at the hips without undue

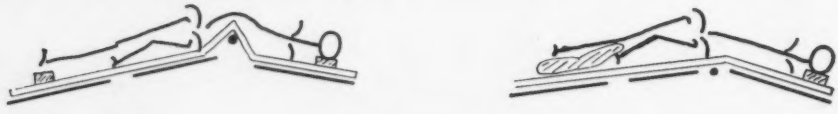

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FIGURE 8. THE KIDNEY POSITION

The patient is positioned with the kidney-lift supporting the crest of the ilium, and a large pillow is placed between the patient's knees, the lower leg being flexed at both the hip and the knee; the operating table is then broken immediately under the flank.

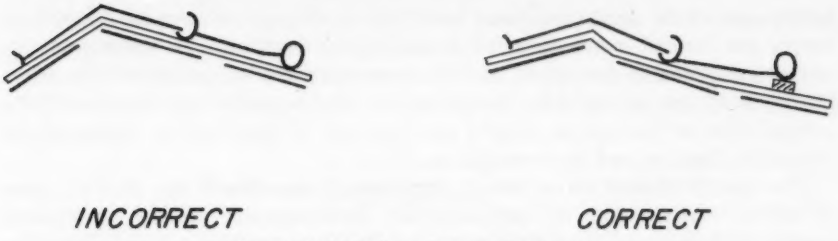

FIGURE 9. THE TRENDELENBURG POSITION

The table is broken to provide flexion at both the hips and the knees, and a pillow is placed under the head to produce flexion at the neck.

strain if at the same time the patient is not allowed to overhang the end of the table too much.

FIGURE 10. THE LITHOTOMY POSITION

The feet or lower legs are padded to prevent pressure from the stirrups; the patient is not allowed to overhang the end of the table; and bolsters or pillows are placed at the side of, and slightly under, the hips.

CONCLUSION

If the respiratory effects of surgical postures are compensated by the use of artificial ventilation; if the circulatory effects are modified by slow and careful movement of the patient, and the avoidance of extreme postures; if the position into which the patient is placed is employed correctly and for as short a time as is surgically expedient—then posture during anaesthesia will be less of a menace to today's patient than it was to John Griffith a century ago.

SUMMARY

The effects of posture on respiration may include changes in pulmonary blood volume, changes in the compliance of the lungs, changes in the intrapulmonary

distribution of the inspired air, and limitations in the expansion of the lungs from simple mechanical interference with the normal diaphragmatic movement, the lateral expansion of the chest, and the anteroposterior expansion of the chest. These effects are reflected by the decreased vital capacity and decreased tidal volume seen in the various surgical postures, and in time lead to fatigue of the respiratory muscles and hypoventilation.

The effects of posture on circulation during anaesthesia are due to vasodilatation, obtundation of compensatory cardiovascular reflexes, peripheral pooling of blood, and, in certain positions, actual obstruction to flow. The deleterious nature of these effects is increased by increased depth of anaesthesia, and is particularly apparent when the position of the patient is changed during anaesthesia.

The effects of posture on the skeletal, muscular, and peripheral nervous systems are due to stresses, strains and stretches of ligaments, muscles, or nerves.

The respiratory depression inherent in many of the surgical postures can be offset by the combined use of an endotracheal tube and assisted or controlled respirations.

The danger of cardiovascular collapse when the patient is moved during anaesthesia can be largely avoided by moving the patient slowly, gently, and smoothly.

The possibility of muscle aches or pains and nerve injuries following the use of the various surgical postures can be lessened by proper use of the position demanded by the specific operative procedure.

Résumé

Les effets de la position sur la respiration peuvent inclure des changements dans le volume sanguin pulmonaire, des changements dans la compliance des poumons, des changements dans la distribution intrapulmonaire de l'air inspiré, des restrictions à l'expansion des poumons par simple nuisance mécanique au mouvement diaphragmatique normal, à l'expansion latérale de la cage thoracique et à l'expression antéro-postérieure du thorax. Ces effets se traduisent par une diminution de la capacité vitale, une diminution de l'air courant observées dans les différentes positions chirurgicales et, avec le temps, conduisent à la fatigue des muscles respiratoires et à l'hypoventilation.

Les effets de la position sur la circulation au cours de l'anesthésie consistent en vasodilatation, dépression des réflexes cardiovasculaires compensateurs, en accumulation périphérique du sang et, dans certaines positions, en arrêt de la circulation locale. La nocivité de ces effets augmente avec la profondeur de l'anesthésie et devient plus manifeste à l'occasion des changements de position du malade au cours de l'anesthésie.

Les effets de la position sur le squelette, les systèmes musculaires et nerveux périphériques sont dus à des tensions, des contusions, des étirements de ligaments, de muscles et de nerfs.

La dépression respiratoire consécutive à plusieurs positions chirurgicales peut être surmontée par l'emploi combiné de l'intubation endotrachéale et de la respiration assistée ou contrôlée. Quant au risque de collapse cardiovasculaire

survenant lorsque l'on bouge le malade sous anesthésie, il est possible de l'éviter en bougeant le malade lentement, gentiment et doucement.

On peut diminuer les douleurs musculaires et les séquelles nerveuses dues aux positions chirurgicales en employant judicieusement la position que requiert l'opération.

REFERENCES

1. Snow, J. On Chloroform and Other Anaesthetics: Their Action and Administration. 1st ed., London: John Churchill (1858).
2. Nicholson, M. J., & Eversole, U. H. Nerve Injuries Incident to Anaesthesia and Operation. Anesth. & Analg. *36*: 19 (1957).
3. Lewis, B. M.; Lin, T. H.; Noe, F. E.; & Komisaruk, R. The Measurement of Pulmonary Capillary Blood Volume and Pulmonary Membrane Diffusing Capacity in Normal Subjects: The Effect sof Exercise and Position. J. Clin. Invest. *37*: 1061 (1958).
4. Sharp, J. T. The Effect of Body Position Change on Lung Compliance in Normal Subjects and in Patients with Congestive Heart Failure. J. Clin. Invest. *38*: 659 (1959).
5. Attinger, E. O., Herschfus, J. A., & Segal, M. The Mechanics of Breathing in Different Body Positions: II, In Cardiopulmonary Disease. J. Clin. Invest. *35*: 912 (1956).
6. Slocum, H. C., Hoeflich, E. A., & Allen, C. R. Circulatory and Respiratory Distress from Extreme Positions on the Operating Table. Surg., Gynec. & Obst. *84*: 1051 (1947).
7. Hamilton, W. F., & Morgan, F. B. Mechanism of the Postural Reduction in Vital Capacity in Relation to Orthopnea and Storage of Blood in the Lungs. Am. J. Physiol. *99*: 526 (1931).
8. McMichael, J., & McGibbon, J. P. Postural Changes in Lung Volume. Clin. Sc. *4*: 175 (1939).
9. Stephen, C. R. The Influence of Posture on Mechanics of Respiration and Vital Capacity. Anesthesiology, *9*: 134 (1948).
10. Sokalchuk, A.; Ellis, D.; Hickcox, C.; & Greisheimer, E. M. Pulmonary Function as Affected by Operative Position. Anesthesiology *10*: 577 (1949).
11. Case, E. H., & Stiles, J. A. The Effect of Various Surgical Positions on the Vital Capacity. Anesthesiology 7: 29 (1946).
12. Jones, J. R., & Jacoby, J. The Effect of Surgical Positions on Respiration. Surg. Forum, V: 686 (1954).
13. Henschel, A. B.; Wyant, G. M.; Dobkin, A. B.; & Henschel, E. O. Posture as It Concerns the Anesthesiologist: A Preliminary Study. Anesth. & Analg. *36*: 69 (1957).
14. Asmussen, E., Christensen, E. H., & Nielson, M. Regulation of Circulation in Different Postures. Surgery *8*: 604 (1940).
15. Peterson, L. H., Eather, K. F., & Dripps, R. D. Postural Changes in the Circulation of Surgical Patients as Studied by a New Method for Recording the Arterial Blood Pressure and Pulse Pressure. Ann. Surg., *131*: 23 (1950).
16. Gordh, T. Postural Circulatory and Respiratory Changes during Ether and Intravenous Anesthesia. Acta chir. scand. *92*: suppl. 102 (1945).
17. Burstein, C. L. Clinical Anesthesia Conference: Positional Hypotension. N.Y. State J. Med. *58*: 4062 (1958).
18. Robertson, J. D., & Swan, A. A. Effect of Anaesthetics on Baroreceptor Reflexes. Q. J. Exp. Physiol. *42*: 113 (1957).
19. Howard, B. K., Goodson, J. D., & Mengert, W. F. Supine Hypotensive Syndrome in Late Pregnancy. Obst. & Gynec. *1*: 371 (1953).
20. Slocum, H. C., O'Neal, K. C., & Allen, C. R. Neurovascular Complications from Malposition on Operating Table. Surg., Gynec. & Obst. *86*: 729 (1948).
21. Wood, D. A. Injuries to Nerves during Anesthesia. Calif. & Western Med. *53*: 267 (1940).
22. Dhunér, K. G. Nerve Injuries following Operations: A Survey of Cases Occurring during a Six-Year Period. Anesthesiology *11*: 289 (1950).

THE EFFECT OF LYSERGIC ACID DIETHYLAMIDE (DELYSID, LSD-25) ON THIOPENTAL RECOVERY

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THE SYNTHESIS of lysergic acid diethylamide by Hofmann (1) and subsequent studies of the effects of this compound in man by Stoll (2) initiated the study of a group of drugs with unique psychotomimetic qualities. LSD-25 in very minute quantities produces psychic effects in man which last twelve hours or more, even though the drug is excreted from the body within one hour (3).

In most normal humans, oral doses of less than 1 μg. per kilogram of LSD-25 produce a syndrome which simulates a moderate acute schizophrenic upheaval with or without catatonic features. These individuals also experience a variety of disturbances of thought processes, depersonalization, hallucinations, abnormal perceptions, delusions, distortions, and misinterpretations (4). These reactions are interpreted in part as an indication of augmentation of perception of dulled areas of the brain (5). Motor effects of LSD-25 include a subjective sense of trembling and objectively an increase in deep reflexes. Sympathetic overactivity is indicated by the appearance of mydriasis and a slight increase in blood pressure and pulse rate (6). The increased sympathetic activity also causes an increase in skin temperature, hyperglycaemia, and pilo-erection. These reactions can be prevented or reversed by chlorpromazine, or general anaesthesia (7).

The hypnotic effect of barbiturates appear to be accompanied by an increased excretion of the major metabolite of 5-hydroxytryptamine (serotonin, 5 HT). In the conscious cat, LSD-25 produces alerting behaviour, and abolishes the E E G spindles of barbiturate anaesthesia (8). Since LSD-25 antagonizes the action of 5 HT in the circulation, one might expect a shortening of the hypnotic action of barbiturates by LSD-25 (9).

Apter showed that LSD-25 was effective in preventing death from lethal doses of pentobarbital (10). However, the effect of LSD-25 against lethal and sub-lethal doses of thiopental was not apparent in dogs, even though the intra-ventricular injection of LSD-25 in the awake dog has a stimulating (analeptic) effect (11).

In view of the potent cortical stimulating effect of LSD-25 in awake humans, the following test was carried out to determine if an analeptic effect against thiopental narcosis could be demonstrated.

METHOD

This study was designed to find out whether LSD-25 shortens the recovery time from thiopental–nitrous oxide anaesthesia for a standard, short operative procedure in women.

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Uterine dilatation and curettage was the procedure selected because the patients' age variation is not great, the posture required is always lithotomy, surgical trauma is very slight, and the physical state is usually good (1 or 2). In order to avoid selection, 55 consecutive patients were used as the control series followed by 55 consecutive patients for the test. The test group received 25 μg. LSD-25 intravenously as soon as the operation and nitrous oxide anaesthesia were discontinued.

At the preoperative visit, the usual assessment of the patient's physical state was made, and 4 to 6 mg. of perphenazine (Trilafon®) was given by mouth at bedtime. Scopolamine 0.4 mg. was given intramuscularly about one hour before the operation. This was the only medication given before anaesthesia. An infusion of 5 per cent dextrose in water was started on each patient to assure rapid absorption of all intravenous drugs. The patient was asked to gaze at the ceiling and to breathe deeply with the mouth open as induction was started with 3ml. of 2.5 per cent thiopental injected quickly. One minute by the clock was then allowed in order to determine the hypnotic response. Addtional thiopental was then injected at a rate of approximately 15 mg./sec., until involuntary closure of the eyelids occurred. A snug-fitting face mask was then applied and nitrous oxide–oxygen (9 L.: 3 L.) was administered from a Mapleson A system (Magill's attachment) with the blow-off valve open on a Boyle or MIE anaesthetic machine until the end of the operation. The flow used in this system was sufficient to prevent longitudinal mixing in the corrugated tube. Immediately after induction the patient was placed in the lithotomy position and preparation was begun for the operation. If the patient showed signs of resistance to stimulation by movements, breath-holding, or swallowing, increments of 50 mg. of thiopental were administered. At least one minute was allowed between added increments, when they were necessary.

Except for the intravenous administration of LSD-25, there were no technical differences in the conduct of anaesthesia in the two groups. Four patients in the control group, and five in the test group were given a small dose of succinylcholine (10–20 mg.) when bimanual pelvic examination proved somewhat difficult because of tight abdominal muscles. This would not affect the duration of anaesthesia, as it was given at the beginning of the procedure.

Recovery time was the time in minutes noted from the moment the operation ended and the mask was removed from the face, until the following four end points were reached and recorded by the nursing staff in the recovery room who were instructed and assigned to observing these patients.

(i) Opened her eyes on request (response to verbal command)
(ii) Answered her name on request (sluggish verbal response)
(iii) Answered her home address on request (alert verbal response)
(iv) Identified simultaneous touch stimulus to face and contralateral arm three times in quick succession (Bender face-hand test)

During the period that these patients were under observation in the recovery room, the vital signs were checked at frequent intervals, and the incidence of vomiting was noted.

Results

The calculated means and standard deviations of the patients' age, height, weight, dose of thiopental (for induction, total, and per kilogram body weight), time of final dose increment (from time of induction), duration of anaesthesia, and times for recovery responses to four end points are summarized in Table I. The Fisher *t* value and significance of the difference of recovery times between the control and test groups are also shown.

It appeared evident from the analysis of these data that LSD-25 in a single intravenous dose of 25 μg. did not exert any analeptic effect which shortened the wakening time from thiopental–nitrous oxide anaesthesia. There was no significant

TABLE I

SUMMARY OF DATA SHOWING THE RECOVERY TIME FROM THIOPENTAL WITHOUT AND WITH LSD-25

				Thiopental					Recovery time in minutes			
Patients	Age	Height (cm.)	Weight (kg.)	Induction dose (mg.)	Total dose (mg.)	Dose/kg. (mg.)	Time of final dose (min.)	Duration of anaesthesia (min.)	R.V.C.	S.V.R.	A.V.R.	B.F. H.T.
Control (55)	40	156	64	353	480	7.5	10.0	19.7	14.3	16.8	17.8	18.4
S.D.±	13	7	15	33				8.1	13.2	13.9	13.8	13.8
LSD-25 (55)	38	155	60	352	444	7.4	9.6	19.4	12.8	16.1	17.3	18.4
S.D.±	13	6	13	29				8.7	10.2	10.8	11.2	11.1
Fisher t value:									0.666	0.295	0.208	0
Probability p:									No significant difference			

R.V.C. = response to verbal command
S.V.R. = sluggish verbal response
A.V.R. = alert verbal response
B.F.H.T. = Bender face-hand test

difference in the blood pressure and pulse rates between the control and test groups in the immediate postoperative period while these patients were under observation in the recovery room. Vomiting occurred in ten of the 110 patients in the recovery room: six of these did not receive LSD-25 and four did. These observations indicate that LSD-25 had no distinguishing cardiovascular effect, nor did it promote vomiting.

DISCUSSION

The comparison of the control and test groups was considered clinically and statistically valid because the age range, size, mean dose of thiopental, duration of the operation, and dose of thiopental per minute of anaesthesia were as similar as is possible to obtain from cases assigned on a surgical schedule.

The clinical method and technique of anaesthesia used was identical to that used by Gale (12), except that the patients in this study were not given a narcotic sedative in their premedication. It was, therefore, no surprise that the mean dose of thiopental and the duration of anaesthesia were comparable to those of the patients in his study who received a "median dose range" of thiopental. The response time to recovery and alertness after anaesthesia were also comparable.

The dose of LSD-25 employed in this study may have been too small to demonstrate an analeptic effect. However, an intravenous dose of 25 μg. LSD-25 is very close to the dose range which causes a variety of disturbances of affective behaviour such as hallucinations, depersonalization, reliving of repressed memories, and other undesirable reactions which may last up to twelve hours. The authors of this study did not feel justified in risking the development of such behaviour merely for the purpose of shortening the drowsy state after anaesthesia. On the other hand, much larger doses of LSD-25 should probably be tested with justification when a patient has taken a very large dose of barbiturates with suicidal intent. If no apparent effect is seen, no harm is done and other forms of therapy may be tried. Recently, one such trial was made on a psychiatric patient in this hospital who had ingested 10 to 15 gm. of Tuinal® with suicidal intent. This

patient was given 400 μg. of LSD-25 orally by the attending physician shortly after he was discovered. He slept deeply for about ten hours, and awakened without ill effects.

It was interesting to note in this study that none of the 55 patients who received LSD-25 had any overt suggestion of the various somatic, mental, or perceptual changes that are observed in subjects who have had LSD-25 without anaesthesia. The authors did not wish to draw the patients' attention to any subjective symptoms that may have occurred; so no attempt was made to elicit data of psychological import. However, the possibility exists that there was an increase in symptom intensity which was not "verbalized," and so could not be specifically observed. Only on the basis of the test data observed may one conclude that LSD-25 has no analeptic action. It may be surmised that the slow release of thiopental from the fat depots into the blood stream over several hours during recovery was sufficient to suppress any overt behavioural changes that may occur even with a small dose of LSD-25.

Summary and Conclusions

Two groups of 55 consecutive female patients were observed closely to determine the recovery time from thiopental–nitrous oxide anaesthesia after a short standard operative procedure. Uterine dilatation and curettage was the operation chosen because it produces very little surgical trauma and requires only a relatively light level of anaesthesia. The first group of patients served as a control. In the second group, each patient received 25 μg. of LSD-25 immediately after the end of anaesthesia.

There was no significant difference in the time required for awakening in the two groups as measured by four end points: response to verbal command (open your eyes), sluggish verbal response (what is your name), alert verbal response (where do you live), and Bender face-hand test (identify simultaneous touch stimulus to face and contralateral arm). There were also no distinguishing cardiovascular or overt psychic changes observed in any of the patients who received LSD-25. It was suggested, however, that one may be justified in testing the effectiveness of a much larger dose of LSD-25 when a very large overdose of a barbiturate has been injected or taken orally with suicidal intent.

Acknowledgment

The authors wish to thank Dr. A. Hoffer of the Department of Psychiatry, University Hospital, Saskatoon, for his help and advice in this study; and Dr. J. M. Grosheintz, Director of Medical Department of Sandoz, Ltd., Montreal, who supplied the D'lysergic acid diethylamide tartrate (Delysid, LSD-25).

Résumé

Nous avons observé de près deux groupes de 55 malades consécutives pour préciser le moment du réveil à la suite d'une anesthésie au thiopenthal et protoxide

d'azote après une courte opération ordinaire. Nous avons choisi comme opération: dilatation et curettage parce que cette opération n'occasionne qu'un très petit traumatisme et ne requiert qu'un niveau d'anesthésie plutôt superficiel.

Le premier groupe de malades nous sert de témoins. Chacune des malades du deuxième groupe a reçu 25 mg. de LSD-25 immédiatement à la fin de l'anesthésie.

Entre les deux groupes, nous n'avons pas observé de différence notable dans la durée du sommeil, évaluée de quatre façons: une réponse à un ordre verbal (ouvrez vos yeux), une réponse verbale marmottée (quel est votre nom), une réponse verbale attentive (quelle est votre adresse), le test Bender (identification simultanée des stimuli du toucher à la face et au bras opposé). Nous n'avons pas observé non plus de changements cardiovasculaires ou d'éveil psychique chez aucune des malades qui avaient reçu LSD-25.

A la lumière de ces tests, nous concluons que LSD-25 est dépourvu d'activité analeptique. Nous concédons, toutefois, que si un grand surdosage de barbituriques a été donné en injection ou a été absorbé par la bouche en tentative de suicide, on puisse tenter de l'employer.

REFERENCES

1. Stoll, A., & Hofmann, A. Partialsynthese von Alkaloiden vom Typus des Ergobasins. Helv. chim. Acta *26*: 944 (1943).
2. Stoll, A. Lysergsaure-diathylamid, ein Phantastikum aus der Mutterkomgruppe. Schweiz f. Arch. Neurol. u. Psychiat. *60*: 279 (1947).
3. Cerletti, A., & Rothlin, E. Role of 5-hydroxytryptamine in Mental Diseases and Its Antagonism to Lysergic Acid Derivatives. Nature *176*: 785 (1955).
4. Rinkel, M.; Hyde, R. W.; Solomon, H. C.; & Hoagland, H. Experimental Psychiatry: II, Clinical and Physio-Chemical Observations in Experimental Psychosis. Am. J. Psychiat. *111*: 881 (1955).
5. Berlin, L.; Guthrie, T.; Weider, A.; Goodell, H.; & Wolff, H. G. Studies in Human Cerebral Function: Effects of Mescaline and Lysergic Acid on Cerebral Processes Pertinent to Creative Activity. J. Nerv. & Ment. Dis. *122*: 487 (1955).
6. Forrer, G. R., & Goldner, R. D. Experimental Physiological Studies with Lysergic Acid Diethylamide (LSD-25). Arch. Neurol. & Psychiat. *65*: 581 (1951).
7. Rothlin, E.; Cerletti, A.; Konzett, H.; Schalch, W. R.; & Taeschler, M. Zentrale vegetative LSD-Effekte. Experientia 12: *154* (1956).
8. Bradley, P. B. Effect of Some Drugs on Electrical Activity of Brain in Cat. Electroencephalog. & Clin. Neurophysiol. *5*: 471 (1953).
9. Brodie, B. B., & Shore, P. A. Concept for Role of Serotonin and Norepinephrine as Chemical Mediators in Brain. Ann. New York Acad. Sc. *66*: 631 (1957).
10. Apter, J. T. Analeptic Action of Lysergic Acid Diethylamide (LSD-25) against Pentobarbital. A.M.A. Arch. Neurol. & Psychiat. *79*: 711 (1958).
11. Dobkin, A. B., & Harland, J. H. Drugs which Stimulate Affective Behaviour: I, Action of LSD-25 against Thiopental in Dogs. Anaesthesia (in press).
12. Gale, A. S. The Effect of Methylphenidate (Ritalin) on Thiopental Recovery. Anesthesiology *19*: 521 (1958).

ELECTROENCEPHALOGRAPHIC MONITORING DURING CARDIAC SURGERY: OBSERVATIONS ON THE EEG RESPONSE TO SEVERE HYPOTENSION

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THE SAFE CONDUCT of anaesthesia requires that the anaesthetist have at all times reliable information concerning the vital functions of his patient. This is generally obtained by observing the usual indices such as blood pressure, pulse, respiration, eye signs etc. In cardiac surgery, however, hypothermia with arrest of the circulation, extracorporeal circulation, sudden accidental hypotension or cardiac arrest, as well as the anaesthetic procedures used for this type of surgery, alter or suppress basic physiological functions and the foregoing indices become uninterpretable. In this situation the anaesthetist can either resign himself to intelligent guesswork or attempt to retain precise control by observing other functions which remain valid and useful under the changed conditions.

To this end, anaesthetists have taken methods from the physiological laboratory into the operating room, for example, arterial O_2 saturation, blood pH, alveolar CO_2, etc.; more recently the development of suitable recording machines has made it possible to monitor the electrocardiogram (ECG) and electroencephalogram (EEG) continuously during surgical operations.

The anaesthetist is particularly interested in the EEG for two reasons: (*a*) it affords an index of the depth of anaesthesia (see review by Wyke (23); and (*b*) it reveals disturbances of cerebral function, especially those due to inadequate cerebral blood flow and to hypoxia which are constant hazards of open heart surgery. For this reason the recording of the EEG has been advocated in the anaesthetic management of cases requiring extracorporeal circulation (13, 18).

To use the EEG efficiently as an indicator of the adequacy of cerebral blood flow and oxygenation, some basic factors have to be considered:

(*a*) Anaesthesia, hypercapnia, hypoxia, hypotension, hypoglycaemia, and hypothermia affect the whole organism and therefore the whole brain. The recording of a single lead is sufficient for practical purposes and a fronto-occipital lead is usually recommended.

(*b*) The response of the EEG to these disturbances is not specific, that is, the observed changes in frequency and amplitude of waves are similar regardless of which disturbance is the cause (4, 17). A more specific diagnosis may be made by elimination, for example, if the depth of anaesthesia remains unchanged and CO_2 accumulation is avoided, then a change in the wave pattern will most likely be due to inadequate oxygenation of the brain caused by either hypoxaemia or an inadequate cerebral blood flow.

(*c*) Any deviation from the normal wave pattern in the EEG can be regarded as a disturbance of the function of brain cells. Cellular function is depressed in two ways:

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(i) By introduction of agents, such as anaesthetics, that depress the function of cerebral cells. The gradual changes in electrical activity which results have been classified according to the depth of anaesthesia (7), and it has been shown that there is a good correlation between the blood level of an anaesthetic agent and the empirically designated EEG level (9). An increase in arterial CO_2 tension appears to have a similar effect (6). The various anaesthetic agents and CO_2 have a summative effect on the EEG, that is, the addition of a second agent will give an EEG pattern similar to that expected from an increase in concentration of the first anaesthetic agent.

(ii) By reduction of the supply of substances essential to normal function. Oxygen and glucose, two most important substances for normal cellular function, have to fall below a critical level (12) before characteristic EEG changes occur. This critical level is considerably below the normal: for example, in the experimental animal a reduction of the O_2 saturation in the jugular vein to 30 per cent (12), or in the arterial blood to 40 per cent (6). During surgery in man, EEG changes have been observed at an arterial O_2 saturation of 70 per cent when produced rapidly (17), and typical EEG signs have been reported in conscious man breathing 100 per cent O_2 at a barometric pressure of 68 mm. Hg. (16) or from mixtures containing 7.8–11.4 per cent O_2 (8). In controlled hypotension in man, no EEG changes were seen above a systolic pressure of 50–60 mm. Hg (15). A hypoglycaemia of 50 mg. per cent was required to produce EEG manifestations (12), but below such a critical level a small further reduction caused marked EEG changes. Disturbances of this type appear to have a summative effect, that is, a moderate hypoxaemia in the presence of a moderate hypotension may result in EEG changes, although either of these disturbances alone would not produce EEG signs. However, anaesthesia and hypoxia do not appear to have a summative effect (17), that is, a greater degree of hypoxia is required to produce recognizable EEG manifestations in deep than in light anaesthesia.

(*d*) Whether a disturbance of the cerebral oxygenation is due to a low O_2 saturation (anoxic anoxia) or to a low blood flow (stagnant hypoxia) cannot be determined accurately at all times under clinical conditions. The EEG response to both conditions is identical, and evidence obtained with both hypoxia and severe hypotension has been used in support of the present interpretations.

Material and Methods

In 25 patients, ranging from 7 to 50 years of age, the EEG (fronto-occipital lead) and the ECG (lead II) were recorded with a direct writing instrument (Schwarzer Cardioscript S4) during surgery. In 13 of these patients open intracardiac operations were done, using a Pump-Oxygenator Model "Mark Cooley." Two other patients were operated under hypothermia. Ten patients underwent a mitral valvulotomy. In all cases of extracorporeal circulation and hypothermia and in three cases of mitral valvulotomy, direct arterial pressures were continuously recorded from the radial artery. The arterial O_2 saturation was determined at intervals using a Kipp oximeter.

Anaesthesia was induced with sodium pentothal and in 22 patients maintained with nitrous oxide: oxygen, Demerol®, or Leritine®. In the remaining three, it was maintained with ether or cyclopropane; d-tubocurare or Anectine were used as relaxants. Respiration was controlled while the thorax was open; a mechanical ventilator (Bennett Assistor) was used in 12 of the patients.

In each patient only the first appearance of EEG changes that were considered due to hypotension has been taken into consideration since subsequent changes coincident with repeated periods of hypotension often require a different interpretation. It was assumed that an arterial O_2 saturation of 90 per cent or more during extracorporeal circulation would exclude hypoxaemia as a cause of the EEG changes observed in these patients.

Observations and Discussion

The EEG Response to Hypotension

A typical EEG response to slowly progressing hypotension (8) is shown in Figure 1; in this patient extracorporeal circulation had been started shortly before. During the period of recording, the blood pressure fell slowly from 44–50 to 35 mm. Hg. With this fall in blood pressure, the normal EEG pattern of light anaesthesia, characterized by fast waves (20–30/sec.) and an amplitude of 30–50 microvolts, as seen in the left upper part of Figure 1, changed into a slow wave pattern with an increased amplitude. These slow waves (5–7/sec.), at first fairly regular, became irregular. Finally very slow, large waves dominated (1–2/sec.) and these persisted until it was possible to raise the blood pressure. A further fall in blood pressure would have caused a reduction in amplitude and

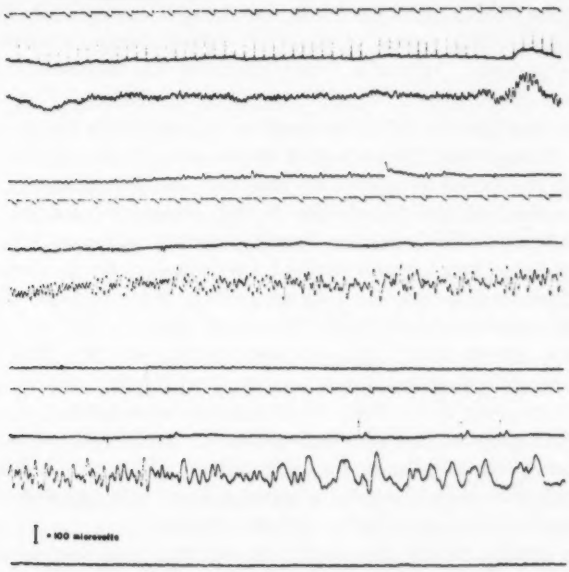

Figure 1. EEG response to hypotension—extracorporeal circulation. Continuous record—*top to bottom*: time marker: 1 sec.; ECG lead II, EEG, arterial blood pressure. *Centre*: Acetylcholine arrest. Note: Change in wave pattern upper right. The occurrence of irregular slow waves of higher amplitude (centre) and large very slow waves (bottom right). A gradual fall in blood pressure can be recognized (upper right side: 46–50 mm. Hg; lower right side: 35 mm. Hg).

more pronounced slowing, leading to a "flattening," that is, a wavy line without any recognizable pattern, and finally a straight line reflecting the disappearance of any cerebral electrical activity (Fig. 3B).

This record shows a close similarity to the EEG changes seen in anaesthesia alone (7). A correlation between the blood level of anaesthetic agent and EEG pattern has been demonstrated (9). It is not possible to correlate blood pressure and EEG changes for the following reasons. The critically low level of blood pressure necessary to produce EEG changes depends not only on individual variations and on age (14) but also on the character of the fall in blood pressure (19). In general, the critical level at which grossly recognizable EEG changes occur is about 50 mm. Hg (4, 15, 19). In this series the first EEG changes were observed between 40 and 60 mm. Hg. A very rapid fall in pressure can produce changes at a higher level (19). With drug-induced hypotension of a slowly progressing type the blood pressure has been observed to fall as low as 36 mm. Hg without causing a change in the EEG (15). In conscious patients, arterial pressures of 29 to 80 mm. Hg have been achieved with hypotensive agents before clinical signs of cerebral anoxia became prominent; the higher any preceding hypertension the higher was the pressure at which the signs appeared (10).

There appears to be a certain degree of adaptation to hypotension as well as hypoxia, presumably brought about by changes in cerebral blood flow and vascular resistance. The return of the EEG pattern towards normal in spite of persistent hypotension (16) or a slowly declining O_2 saturation (18) has been observed.

The failure of supply of O_2 to the brain is the critical factor in the production of the EEG changes that are important to the anaesthetist. If the oxygen-carrying power of the blood is adequate, and this can be measured or estimated reliably, then the cerebral blood flow is the important variable. Therefore, it is very likely that the EEG changes would correlate better with a critically low blood flow than with a certain level of hypotension. Such a relationship was established for clinical signs of hypoxia which were seen to appear with a cerebral blood flow of about 30 ml./100 cc. of brain (10, 21).

The additive effects of hypotension and hypoxia on the EEG were demonstrated by one of our patients. Large slow waves were observed at a blood pressure of 70 mm. Hg about three minutes after the beginning of extracorporeal circulation. An immediate sample showed that the arterial O_2 saturation was only 69 per cent and subsequently an insufficient flow of O_2 to the bubble oxygenator was found and corrected. About three minutes later, although the blood pressure had not changed, the normal EEG activity returned.

A response similar to the one shown in Figure 1 was seen in twelve of our patients. With one exception, the EEG changes were always related to a critically low blood pressure. Severe hypotension always developed shortly after the beginning of extracorporeal circulation. Even with a low-flow technique, as in our cases, the blood pressure usually stabilized and often rose after about three to five minutes although the output of the pump-oxygenator was not increased. This stabilization or rise probably represents a compensatory adjust-

ment of the patient's peripheral circulation. The EEG usually reflected these falls of arterial pressure below the critical level for adequate cerebral circulation. In the one patient in whom the blood pressure did not fall below 55 mm. Hg, the EEG remained unchanged.

It has been thought that the perfusion of the brain with blood at room temperature is responsible for a transient slow wave pattern which appears shortly after the beginning of extracorporeal circulation (22). The present observations do not support such a view, the appearance of slow waves was always associated with a critically low arterial pressure.

A somewhat different response of the EEG to hypotension has been occasionally observed. In two cases of valvulotomy, although slow waves did not appear, there was a rapid decrease in amplitude and a temporary flattening of the EEG (Fig. 2).

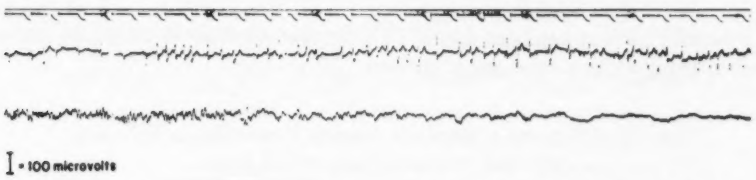

FIGURE 2. Mitral stenosis: rapid depression of amplitude and flattening of EEG without distinct changes in frequency.

A decrease in amplitude of the EEG without significant slowing has been considered to be an early response to hypoxia (17) and has also been the predominant sign observed in a study of drug-induced hypotension (15). Under these conditions this was succeeded by a slow wave pattern. On the other hand, others have observed a depression of amplitude and flattening without the appearance of distinct slow waves following total occlusion of the blood supply to the brain (2) or cardiac asystole (1). Anoxia in rats, produced by rapid decompression, also produced a decrease in voltage and flattening of the EEG without the appearance of slow waves (20). Schallek and Walz (19) were able to demonstrate that the speed of development of hypotension can determine the response: a sudden fall in pressure produced flattening immediately; a gradual fall produced slow waves first.

The following observations underline this importance of the suddenness of onset of hypotension to the EEG response. A rather rapid fall in blood pressure shortly after the beginning of extracorporeal circulation produced slow waves (Fig. 3A) similar to those shown in Figure 1, but only briefly. Occlusion of the cardiac inflow under hypothermia produced an even briefer episode of slow waves with only a slight increase in voltage, followed by flattening and electrical silence (Fig. 3B). Sudden cessation of the circulation brought about by the surgeon's finger during a mitral valvulotomy, resulted in a depression of the EEG without slow waves (Fig. 3C).

This latter response (Figs. 2, 3C) has always been observed in patients or experimental animals under anaesthesia; only one report of similar EEG changes

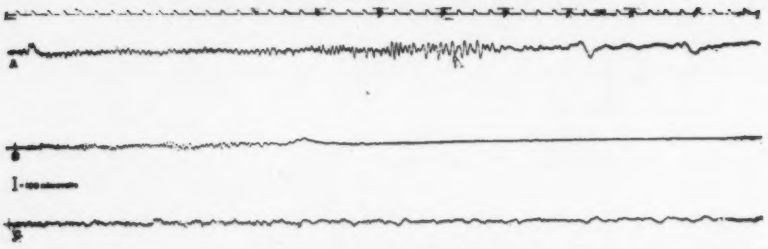

FIGURE 3. EEG response to reduction of cerebral bloodflow. *Top* (A): moderately rapid blood pressure fall during extracorporeal circulation; note the distinct slow wave pattern preceding flattening of the EEG. *Middle* (B): interruption of cardiac inflow under hypothermia (31° C.). *Bottom* (C): sudden extreme hypotension during mitral valvulotomy. Slow wave pattern absent. Time marker: 1 sec.

in a conscious patient has been found (3). Cardiac asystole in conscious patients, however, resulted in a slow wave pattern (12).

THE TIME RELATION BETWEEN SUDDEN HYPOTENSIVE HYPOXIA AND THE APPEARANCE OF EEG SIGNS

To demonstrate the time relation between the onset of severe hypotension and/or hypoxia, a record of the ECG, EEG, and blood pressure (radial artery) during a mitral valvulotomy is presented (Fig. 4). The entrance of the surgeon's finger into the mitral orifice was marked by the appearance of extrasystoles and a sharp fall in blood pressure. About ten seconds after the beginning of the hypotensive period, large slow waves appeared in the EEG, the blood pressure returned to normal, and only eight seconds later a normal EEG pattern had been restored. A second hypotensive episode was interrupted after seven seconds by three beats with a normal blood pressure, and no definite EEG changes appeared. This brief restoration of blood flow was apparently adequate to prevent EEG changes for another ten seconds. Then the large slow waves reappeared only to disappear when the blood pressure was eventually restored.

Normally the brain contains at any given moment about 7 ml. of oxygen which are used up in about ten seconds (14). Sudden cessation of the heartbeat due to vagal stimulation produced EEG changes only after the time equivalent of at least seven beats had elapsed (12). A time interval of four to six seconds was seen in man between the drop in arterial oxygen reflected in the Ear Oximeter and EEG changes during experimental hypoxia (17). This slight delay in the effect of hypoxaemia on the EEG is similar to that observed in the foregoing clinical case.

Theoretically, breathing of pure O_2 before the onset of a critically low blood pressure should delay the appearance of EEG signs for only a few seconds. A moderate degree of hypoxia before the onset of hypotension should hasten the onset of EEG changes.

For practical purposes such a time lag is of very little significance, and it can be stated that a fall of blood pressure below a critical level is almost immediately

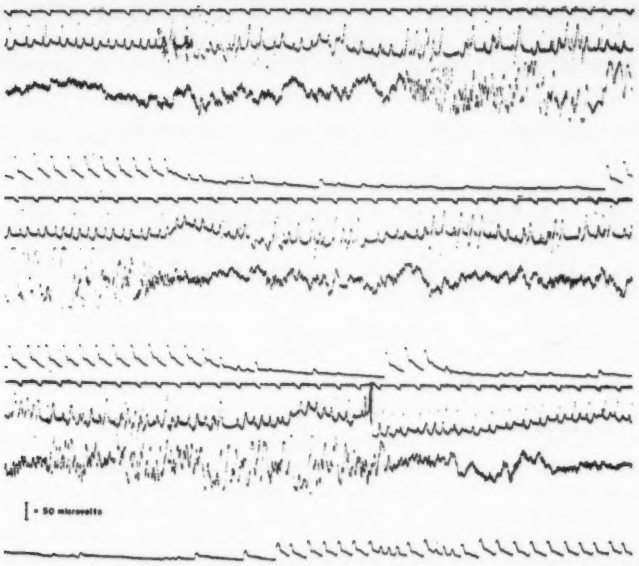

FIGURE 4. Mitral valvulotomy—continuous record. *Top to bottom*: time marker 1 sec., ECG, EEG, arterial pressure. Occlusion of mitral orifice by surgeon results in sudden hypotension which is followed by EEG changes (top) within 10 sec. The persistence of pulse waves indicates that there is still some blood flow. Return of blood pressure reverts EEG changes completely.

reflected in the EEG. EEG changes can apparently be delayed, at least temporarily, however, by an intermittent although brief restoration of blood flow.

CEREBRAL AND CARDIAC TOLERANCE TO HYPOTENSION

It is reasonable to assume that the appearance of EEG changes during a period of hypotension signifies a disturbance in cerebral function due to hypotensive hypoxia. The absence of abnormal changes during hypotension provides reasonable evidence that the brain can tolerate the existing hypotension without ill effect. The question arises whether or not the rest of the organism can also tolerate such a hypotension.

The only other organ in which acute disturbances in function can be detected is the heart. In acute fatal anoxia in animals, it was always found that EEG changes occurred much earlier than ECG changes (20). This could mean either that the time lag after the onset of the critical degree of hypoxia or hypotension is greater for the heart than for the brain, or that the heart can tolerate a greater degree of these deficits without embarrassment. In general, the heart appears less sensitive than the brain to hypoxia.

Although this may be true for the normal heart, the diseased heart may be more vulnerable. This is demonstrated by the record of a patient with a grade

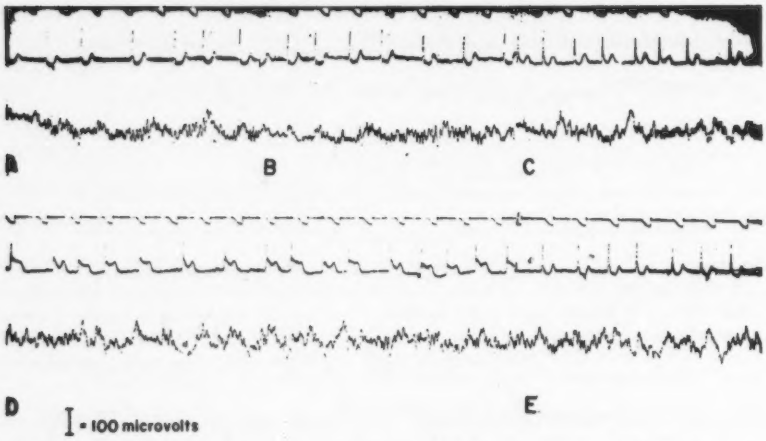

FIGURE 5. ECG changes indicating myocardial hypoxia without EEG changes. (Mitral valvulotomy). *Top to bottom*: time marker (1 sec.) ECG, EEG. A Immediately before valvulotomy. B. Approximately 1 min. after completion of intracardiac procedure, blood pressure 60 mm. Hg. C. 2 min. after B, blood pressure approximately 60 mm. Hg; slight elevation ST segment. D. 2 min. after C, blood pressure unchanged, marked elevation ST segment. E. 3 min. after D, 10 mg. Vasoxyl was given intravenously 2 min. before; blood pressure 100 mm. Hg.

III mitral stenosis (Fig. 5). Following the valvulotomy in this case the blood pressure remained at about 60 mm. Hg systolic (indirect). During this period, no significant EEG changes were seen, but ECG signs of myocardial hypoxia developed (Fig. 5C, D); the high elevation of the ST segment disappeared promptly on the restoration of a normal blood pressure (Fig. 5E).

Despite the usual greater sensitivity of the brain (EEG) to hypotensive and other forms of hypoxia, one should not conclude that other organs, particularly if they are diseased, are always safe if cerebral electrical activity is maintained.

CONCLUSION AND SUMMARY

The electrical activity of the brain represents a function of the cortex and changes of these cortical potentials or their disappearance indicate a disturbance severe enough to alter or abolish the function of cerebral cells. Changes of the normal EEG pattern occur when the O_2 supply to the brain has reached a critically low level, owing to either an insufficient blood flow or a low 0_2 saturation. Although several factors can produce a similar sequence of events in the EEG, it is possible in most instances to exclude a number of these variables and thus determine the most likely cause for the altered EEG pattern.

In cardiac surgery, where episodes of reduced blood flow frequently occur, the EEG is a useful instrument to warn the anaesthetist that a vital function is threatened, providing its limitations, which have been discussed, are taken into account. This warning comes late; only a severe degree of hypotension or hypoxia

will produce EEG manifestations. But these signs occur some time before it is too late to prevent irreversible damage and they may be the only indication to the anaesthetist of the critical state of his patient. During extracorporeal circulation, where many of the usual vital signs fail, the EEG appears to be the most valuable indicator of an adequate cerebral circulation, which can be readily observed and recorded.

In the presented series of cases, two types of EEG responses to hypotension have been observed: (i) a pattern of large slow waves, leading to depression of the amplitude and flattening of the EEG; and (ii) a rapid depression of the normal waves, leading to flattening without the appearance of slow waves.

Observations have been presented which seem to indicate that the suddenness of onset of hypoxia or hypotension is responsible for the type of response. There may also be a difference depending on whether such a disturbance occurs in a conscious or anaesthetized patient.

The sequence of events during a mitral valvulotomy was used to demonstrate the time relation between the onset of hypotension and the appearance of EEG signs. The delay of about ten seconds is probably determined by the circulation time and by the existing state of oxygenation.

During a period of hypotension in a patient undergoing a mitral valvulotomy electrocardiographic signs of myocardial ischaemia were seen without EEG changes. Although the brain is usually more sensitive to hypoxia than the heart, this observation demonstrates the markedly reduced tolerance to hypotension of the diseased heart. It is, therefore, concluded that a degree of hypotension that fails to produce EEG changes cannot generally be regarded as safe for the whole organism.

Acknowledgments

The author wishes to express his thanks to Professor J. A. F. Stevenson, Head of the Department of Physiology, University of Western Ontario, for his encouragement and constructive criticism, to Victoria Hospital (Superintendent Dr. C. J. Kirk) for the provision of the equipment, and to Mr. B. Forbes for technical assistance. The co-operation of Dr. J. C. Coles, Department of Surgery, and the members of the Department of Anaesthesia, was much appreciated.

Résumé

L'activité électrique du cerveau atteste une fonction corticale et les changements de ces potentiels corticaux ou leur disparition indiquent des perturbations assez importantes pour modifier ou abolir la fonction des cellules cérébrales. Il apparaît des changements au tracé normal électroencéphalographique, lorsque l'apport d'oxygène au cerveau a atteint un niveau très bas, soit à cause d'une circulation sanguine insuffisante, soit à cause d'une teneur trop faible en oxygène. Bien que plusieurs facteurs peuvent produire les mêmes modifications sur le tracé électroencéphalographique, il est possible, dans la plupart des cas, d'éliminer un certain nombre de ces facteurs et d'en venir à préciser ce qui, selon toute vraisemblance, produit les modifications sur le tracé.

Au cours de la chirurgie cardiaque, des épisodes de ralentissement circulatoire s'observent fréquemment, alors le tracé électroencéphalographique devient un instrument précieux pour avertir l'anesthésiste lorsqu'une fonction vitale est menacée jusqu'à un certain point. Cet avertissement survient tardivement car seulement une hypotension ou une hypoxie sérieuse peut produire des modifications sur le tracé électroencéphalographique. Mais il arrive que ces signes apparaissent avant qu'il ne soit trop tard pour prévenir l'apparition d'une lésion irréversible ou encore ils peuvent devenir la seule indication pour l'anesthésiste que son malade est dans un état critique. Au cours de la circulation extracorporelle, où plusieurs signes vitaux habituels sont absents, le tracé électroencéphalographique semble être le plus précieux indice d'une circulation cérébrale adéquate, indice que l'on peut observer à tous les instants et enregistrer.

Au cours des séries de cas que nous présentons, nous avons noté deux types de tracés électroencéphalographiques en réponse à de l'hypotension: (i) un tracé montrant de grandes ondes lentes, conduisant à une diminution d'amplitude et à un aplatissement du tracé, et (ii) une dépression rapide des ondes normales, conduisant à l'aplatissement du tracé sans l'apparition d'ondes lentes.

Nous avons présenté des cas qui semblent indiquer que la vitesse d'apparition de l'hypoxie ou de l'hypotension est responsable du type de réponse. Il peut également exister une différence selon que le malade est conscient ou sans anesthésie.

Nous avons employé l'ordre des gestes opératoires au cours d'une valvulotomie mitrale pour illustrer la relation qu'il existe entre l'apparition de l'hypotension et l'apparition des signes électroencéphalographiques. On peut fixer un délai d'environ dix secondes à cause du temps circulatoire et de l'état d'oxygénation.

Au cours d'un moment d'hypotension, chez un malade subissant une valvulotomie mitrale, on a noté des signes électrocardiographiques d'ischémie du myocarde sans observer de changements sur le tracé électroencéphalographique. En dépit du fait que le cerveau soit habituellement plus sensible que le cœur à l'hypoxie, cette observation démontre combien réduite est la tolérance à l'hypotension chez un cœur malade. Nous concluons, en conséquence, qu'un degré d'hypotension qui ne parvient pas à produire des modifications du tracé électroencéphalographique, ne peut pas, en général, être envisagé de toute sécurité pour tout l'organisme.

REFERENCES

1. Ajmone-Marsau, C., & Fuortes, G. F. Electrographic Study of the Convulsant Action of Intravenously Administered Acetylcholine. EEG & Clin. Neurophysiol. *1*:283 (1949).
2. Asenjo, A. Ueber die Wirkung des Extracranialen Verschlusses der Hirngefässe auf die Bioelektrische Tätigkeit der Hirnrinde, Zbl. f. Neurochirurgie *4*:41 (1939).
3. Aymes, E. W., Mahony, D. V., & Goodman, R. D. Adam-Stokes Syndrome and Cerebral Anoxia, J. Nerv. & Ment. Dis. *117*:334 (1953).
4. Beecher, H. K., McDonough, F. K., & Forbes, A. Effect of Blood Pressure Changes on Cortical Potentials during Anaesthesia. J. Neurophysiol. *1*:324 (1938).
5. Bromage, P. R. Some Electroencephalographic Changes Associated with Induced Vascular Hypotension., Proc. Roy. Soc. Med. *46*:919 (1953).

6. Clowes, G. H. A., Jr.; Ketchmer, H. E.; McBurnex, R. W.; & Simeone, F. A. The EEG in the Evaluation of Anaesthetic Agents and Carbon Dioxide Accumulation during Surgery. Ann. Surg. *138*:558 (1953).
7. Courtin, R. F., Bickford, R. G., & Faulconer, A., Jr. The Classification and Significance of Electro-Encephalographic Patterns Produced by Nitrous-Oxide–Ether Anaesthesia during Surgical Operations. Proc. Staff Meet. Mayo Clinic *25*:197 (1950).
8. Davis, P. A., Davis, H., & Thompson, J. W. Progressive Changes in the Human EEG under Low O_2 Tensions. Am. J. Physiol. *123*:51 (1938).
9. Faulconer, A., Jr. Correlation of Concentration of Ether in Arterial Blood with Electroencephalographic Patterns Occurring during Ether-Oxygen and during Nitrous Oxide, Ether and Oxygen Anaesthesia in Human Surgical Patients. Anesthesiology *13*:361 (1952).
10. Finnerty, F. A., Jr., Witkin, Lloyd, & Fayekas, J. F. Cerebral Hemodynamics during Cerebral Ischemia Induced by Acute Hypotension. J. Clin. Invest. *33*:1227 (1954).
11. Gastaut, H., & Fischer-Williams, M. Electro-Encephalographic Study of Syncope. Lancet II:1018 (1951).
12. Gibbs, F. A., William, D., & Gibbs, E. L. Modification of the Cortical Frequence Spectrum by Changes in CO_2, Bloodsugar and O_2. J. Neurophysiol. *3*:49 (1939).
13. Hale, D. E., & Moraca, P. P. Electrocardiogram and Electroencephalogram in Elective Cardiac Arrest. J.A.M.A. *166*:1672 (1958).
14. Kety, S. S. Circulation and Metabolism of the Human Brain in Health and Disease. Am. J. Medicine *8*:205 (1950).
15. Kubicki, St., & Just, O. Das hirnelektrische Bild bei extremer künstlicher Blutdrucksenkung. Der Anestsetist *6*:143 (1957).
16. Luft, U. C., & Noell, W. K. The Manifestations of Sudden Brief Anoxia in Man. Rep. #55–86, School of Aviation Medicine USA, Randolph AFB, Texas.
17. Noell, W. K., Brown, E. S., & Elam, J. O. The Detection of Impairment of Oxygenation: Encephalography and Oximetry during Surgery. Rep. #2, USAF School of Aviation Medicine, Randolph Field, Texas (1954).
18. Patrick, R. T., Theye, R. A., & Moffit, E. A. Studies in Extracorporeal Circulation: V, Anaesthesia and Supportive Care during Intracardiac Surgery with the Gibbon Type Pump Oxigenator. Anesthesiology *18*:673 (1957).
19. Schallek, W., & Walz, D. Effect of Drug Induced Hypotension on the Encephalogram of the Dog. Anesthesiology *15*:673 (1954).
20. Spoerel, W. E. Unpublished data.
21. Stevens, H., & Fazekas, J. F. Experimentally Induced Hypotension: Clinical and Electroencephalographic Consequences. Arch. Neurol. Psychiat. *73*:416 (1955).
22. Theye, R. A., Patrick, R. T., & Kirklin, J. W. The EEG in Patients Undergoing Open Intracardiac Operations with the Aid of Extracorporeal Circulation. J. Thorac. Surg. *34*:709 (1957).
23. Wyke, B. D. Electrographic Monitoring of Anaesthesia. Brit. Med. Bull. *14*:58 (1958).

THE ANALGESIC PROPERTIES OF ANILERIDINE, MEPERIDINE, AND MORPHINE: A COMPARATIVE STUDY[1]

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AND C. W. P. LUNDERVILLE, B.SC., M.D., C.M.[2]

THE EVALUATION of a new drug requires that its effects be tested under controlled conditions in large numbers of clinical cases. This is particularly true of analgesic drugs since none of the methods proposed for objective measurement of the value of these drugs in man can be considered wholly satisfactory.

Although the analgesic effectiveness of anileridine (Leritine®) has been previously recorded by a number of investigators (1–5), we believe that the addition of yet another study to this series may help to orientate the usefulness of this drug among the established analgesic agents. We have, therefore, compared the analgesic activity of anileridine with the effectiveness of morphia and meperidine in a series of unselected patients in the immediate postoperative period.

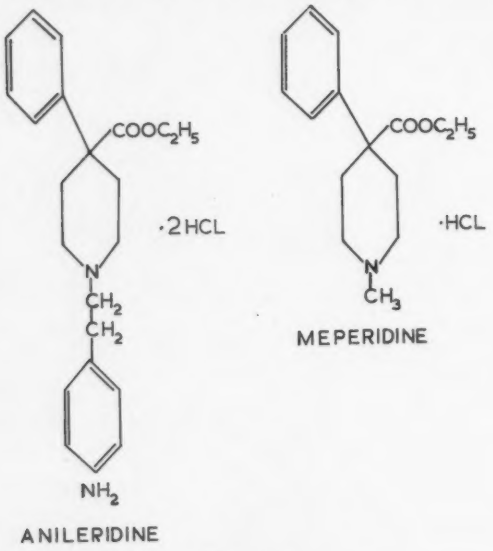

FIGURE 1

Anileridine resembles meperidine in chemical structure. The two drugs are represented by the formulae in Figure 1. Anileridine was first described by Weijlard *et al.* (6) in May 1956.

[1]Presented at the Annual Meeting, Canadian Anaesthetists Society, May, 1959.
[2]Department of Anaesthesia, University of Toronto and the Toronto General Hospital.

Method and Material

Anileridine, meperidine, and morphine were administered to unselected postoperative patients in the recovery room in a blind study. Stable solutions of each drug were prepared in identical multiple dose bottles, each containing 30 cc. Each bottle was labelled by a code number only, and the code was not broken until the study had been completed. Since earlier reports had indicated that the analgesic potency of anileridine is approximately twice that of meperidine, and that of meperidine approximately one-tenth of morphine, weight for weight, the solutions were made to contain:

Anileridine	25 mg. per cc.
Meperidine	50 mg. per cc.
Morphine	5 mg. per cc.

These drugs were administered by experienced recovery room nurses who have for some years judged the requirement of postoperative patients for sedative. We consider these nurses to be our most reliable observers in this matter. The information supplied to the nursing staff was simply that each cubic centimetre of solution was equivalent to 50 mg. of meperidine.

The drugs were administered by intramuscular injection, and blood pressure, pulse, and respiratory rate were recorded at the time of administration. The administration of an analgesic drug was indicated in each case by complaint of pain, or by restlessness judged to be produced by pain. Effect of the drug in the dose given, blood pressure, pulse rate, and respiratory rate were recorded thirty minutes after administration. Patients requiring further sedation in the recovery room received the same code number as the original dose, except for a few cases in which the recovery room staff failed to carry out this plan. Time elapsed from the last administration of sedative in the recovery room to the first sedative required after return to the floor was recorded from the patients' records on the following day.

In all, 1,117 patients are included in this study. The distribution of these patients by drug given is shown below:

Anileridine	382
Meperidine	389
Morphine	346
	1,117

Age Distribution

The youngest patient in this series was 15 years of age, and the oldest 86 years. Age distribution of patients receiving each drug is represented in Table I. It is so similar in each instance that it can play no significant part in the assessment of the results.

TABLE I

	Age group (years)			
	15–29	30–49	50–69	70+
Anileridine	63	151	133	35
Meperidine	57	176	121	35
Morphine	56	156	103	31

Operative Site

It is generally recognized that the operative site greatly influences the requirement of the patient for sedative. We have, therefore, examined the distribution of operative site in this series and this is represented for each drug in Table II. The full range of general surgery and surgical specialties has been abundantly covered, with the exception of cardiac surgery. Here again we find that in most instances the distribution for each drug is sufficiently similar that this factor is unlikely to influence the assessment of the effectiveness of drugs in the comparison.

TABLE II

	Anileridine	Meperidine	Morphine
Intracranial	—	—	2
Head and neck	24	20	26
Thoracic	12	8	12
Abdominal			
Upper	63	54	53
Lower	97	114	78
Perineal	42	42	33
Transurethral	21	25	27
Plastic	13	12	20
Extremities	70	59	54
Back	25	36	31
Breast	15	19	10

Average Dose

In recording the dosage of drugs in this series, simple notation of the number of cubic centimetres given was made in the record. It is of interest in examining these records that the nursing staff quickly established an average effective dose, and that this varied from 1 to 2 cc. of the coded solutions, the dose being based on the assessment of the status of the patient and the individual requirement for sedative. It has been of interest to determine the comparative average dose of these three drugs since we feel that this gives some expression of the relative potency of the preparations. Average dose in milligrams of the three drugs for the patients in the series is shown below:

Anileridine	38.8 mg.
Meperidine	73.6 mg.
Morphine	8 mg.

Sedative Effect

The sedative effect of the drugs was judged by the recovery room nurses at a period of thirty minutes after intramuscular administration. The assessments recorded for each dose are shown in Table III. A good effect was one in which it was evident that no further sedative was required at the end of thirty minutes, a fair effect was one in which relief was probably adequate, but left something to be desired, while a poor effect is recorded in the patient who required additional sedative after the first thirty minutes. It is interesting to note that, in the group having a poor effect from the sedative, there are several who are reported to have had a poor effect from additional sedative. It is probable that, in some of these cases, some other factor was contributing to restlessness.

TABLE III

	No. of patients	No. of doses	Sedative effect		
			Good	Fair	Poor
Anileridine	382	392	350	38	4
Meperidine	389	403	343	45	13
Morphine	346	349	265	64	18

It is of interest to note that there are more good and fair effects recorded for anileridine and meperidine than for morphia. We feel that this firmly establishes the analgesic value of anileridine, and we believe that the differences shown in Table III between anileridine and meperidine are not significant in a study of this kind. It is possible that the potency of anileridine is something greater than twice that of meperidine, so that the standard solutions used in this study did not actually represent equipotent doses, volume for volume. In this regard, it is of interest that Dripps and his collaborators in a similar study (3) calculated that 40 mg. of anileridine was equivalent to 100 mg. of meperidine in analgesic potency. Our study does not permit an accurate calculation of this kind, but the results shown in Table III might tend to bear out this assumption.

It might also be suggested from the data shown in Table III that the generally accepted therapeutic equivalents of 10 mg. of morphine and 100 mg. of meperidine may not be quite accurate, but that meperidine is somewhat more potent than this in relation to morphine.

Duration of Sedative Action

An attempt was made to compare the duration of sedative action of the three drugs by recording the time from the last administration of sedative in the recovery room to the next sedative required by each patient after return to the ward. The time of the first sedative on the ward was taken from the patient's record on the day following operation. The data so derived are shown in Table IV.

TABLE IV

	Time to next sedative (hours)		
	Shortest	Longest	Average
Anileridine	0.5	17	5.4
Meperidine	0.5	17	4.73
Morphine	0.5	20	5.46

There appears to be no significant difference between the three drugs. We are aware that these figures may be open to question since we were unable to control the administration of the sedative on the floor, and we are aware that it is a habit of some nurses to administer an analgesic to every postoperative patient immediately he comes under their care. We do feel, however, that we may assume from these figures that the three drugs have approximately the same duration of action.

A number of patients required no further sedative after leaving the recovery

room. Of these, 43 had been given anileridine, 39 had received meperidine, and 48 had received morphine.

Cardiovascular Effects

As we have previously noted, blood pressure and pulse rate were recorded at the time of administration of the analgesic drug, and after thirty minutes. It is interesting that there is no consistent pattern in the changes which were recorded after the administration of sedative drugs. Blood pressure increased by 10 mm. of mercury systolic as frequently as it decreased by the same amount, and in all but two patients remained within normal limits for the patient at that time. In the patients who received anileridine, blood pressure rose ten or more points on 78 occasions, and was reduced more than 10 mm. of mercury systolic on 91 occasions. In the morphine series, blood pressure rose on 95 occasions and was reduced in 85 patients. In the patients receiving meperidine, blood pressure rose on 75 occasions and was reduced on 99 occasions. Only two patients in this series had declines of blood pressure following the administration of an analgesic drug which caused any concern. Both the patients had received meperidine. The first of these was a postoperative mastectomy whose blood pressure dropped to 75 mm. of mercury within thirty minutes of receiving the sedative. Someone ordered Lorfan® for this patient, although there is no record that the respiration had shown any change, and her blood pressure promptly rose to 100 mm. Hg systolic. The second patient had a cholecystectomy and within the thirty-minute period after receiving meperidine had a drop of blood pressure to 60 mm. mercury. This patient received a vasopressor drug, the foot of the bed was elevated, and the blood pressure returned to normal limits.

There were no cases of circulatory depression in the anileridine series or the morphine series.

We have been interested in the fact that in many cases blood pressure rose following the administration of analgesic drugs. We feel that the changes in blood pressure have been related rather to the relief of pain than to specific cardiovascular effects of the drugs in the doses used in this series. We have examined these changes in blood pressure in relation to operative site, and it is apparent that there is an outstanding tendency for the pressure to rise following the use of analgesics in patients who have had abdominal operations, and a tendency for it to fall from higher than normal to normal levels in other patients. We would infer from this experience that the original pressure levels have been influenced by the stimulus of pain, and that there has been a tendency for the pressure to return towards the patient's normal level when the pain was relieved

Effect on Respiration

Depression of respiration is a pharmacological property which is common to all three of the drugs in this comparison. We were interested, therefore, to record changes in respiration in the patients of this series. As our criterion of a significant change, we adopted an increase or decrease of respiratory rate of four or more breaths per minute. We were interested to find that respiration increased almost as often as it decreased following the administration of these drugs. In nearly every

instance where respiration was decreased, the decrease represented a change from a faster than normal respiratory rate to a rate in a more normal range. One patient who received anileridine had a decrease from 18 per minute to 14 per minute. Two patients who received meperidine had a similar decrease while two patients who received morphine had respiratory rates decreased from 20 per minute to 16 per minute. There was no case of marked respiratory depression in this series.

Discussion

It is evident from examination of the records of this series of 1,117 patients who have received anileridine, meperidine, or morphine for the relief of post-operative pain that all three drugs will produce satisfactory analgesia in doses which do not produce any significant change in cardiovascular or respiratory function. Anileridine (Leritine®) has been found to be a most satisfactory analgesic drug with a therapeutic potency somewhat greater than twice that of meperidine. It has been found satisfactory in all age groups for the relief of pain in the postoperative period.

Aknowledgments

We wish to acknowledge the invaluable help of Mrs. Isabel Corrigan, R.N., Mrs. Christina Jeffries, R.N., and the recovery room staff of the Toronto General Hospital, without whose invaluable assistance this study could not have been undertaken.

This study was financed by a grant-in-aid from Merck and Company Canada Limited.

Résumé

Au cours d'une étude avec des produits non identifiés, nous avons administré dans les suites opératoires, à la salle de réveil, à des malades non choisis, de l'anileridine, de la mépéridine et de la morphine. Chacun des médicaments était préparé en solution stable dans des bouteilles identiques à doses multiples et chacune de ces bouteilles ne portait qu'un chiffre correspondant à une légende. Les solutions étaient ainsi préparées que chaque ml contenait soit l'Anileridine (Leritine®) 25 mg., soit mépéridine (Demerol®) 50 mg., soit morphine 5 mg. Ces médicaments ont été administrés par des garde-malades d'expérience dans la salle de réveil qui n'avaient, comme seule information, que chaque millilitre de solution équivalait à 50 mg. de mépéridine. On a noté, trente minutes après l'administration du médicament, l'effet du médicament à la dose donnée sur la tension sanguine, le rythme cardiaque et respiratoire. Le lendemain, on a noté également le temps écoulé entre le moment de la dernière injection à la salle de réveil et le moment de la première sédation à la chambre du malade.

Cette étude a été faite sur 1,117 malades. Le premier tableau nous fait voir le partage des malades selon le médicament donné. En ce qui concerne l'âge et le site opératoire, le partage des trois médicaments a été semblable. Les chiffres au page 33 nous font connaître la dose moyenne de chaque médicament au cours de l'étude.

L'effet sédatif de ces médicaments a été apprécié par les garde-malades de la salle de réveil, trente minutes après leur injection intramusculaire. Nous voyons dans le tableau III le résultat de chacune des injections. Les effets jugés "bons" et "passables" sont plus nombreux pour l'anileridine et la mépéridine que pour la morphine. Cela est fortement en faveur de la valeur analgésique de l'anileridine Cela nous fait croire également que les équivalences thérapeutiques généralement acceptées, de 10 mg. de morphine et de 100 mg. de mépéridine, peuvent bien ne pas être aussi exactes que nous le croyons.

En ce qui concerne la durée d'action, il ne semble pas exister de différence importante entre les trois médicaments. Nous n'avons pas observé d'effets cardio-vasculaires particuliers chez aucun malade et, au cours de cette étude, aux doses employées, nous n'avons noté aucune dépression respiratoire. L'anileridine (Leritine®) s'est avérée une médication analgésique des plus satisfaisantes.

REFERENCES

1. Orahovats, P. D., Lehman, E. G., & Chapin, E. W. Pharmacology of ethyl-1-(4-amino phenethyl)-4-phenylisonopecatate, Anileridine: A New Potent Synthetic Analgesic. J. Pharmacol. & Exper. Therap. *119*:26–34 (Jan. 1957).
2. Chang, F. F. C., Sofar, P., & Lasagna, L. Comparison of the Analgesic Potency and Side Effects of Anileridine and Demerol in Man. Fed. Proc. 16:288 (March 1957).
3. Dripps, R. D., Millar, R. A., & Kneale, D. H. A Comparison of Anileridine, Morphine and Meperidine in Man. Surg., Gynec. & Obst. *105*:322–326 (Sept. 1957).
4. Keats, A. S., Telford, J., & Kurosu, Y. Studies of Analgesic Drugs: Anileridine Dihydrochloride. Anesthesiology *18*:690–697 (Sept.–Oct. 1957).
5. Wallenstein, S. L., & Houde, R. W. Clinical Evaluation of Relative Analgesic Potencies of Anileridine, Meperidine and Morphine. J. Pharmacol. & Exper. Therap. *122*:81A (Jan. 1958).
6. Weiljlard, J.; Orahovats, P. D.; Sullivan, A. P., Jr.; Purdue, G.; Heath, F. K.; & Pfister, K., III. A New Synthetic Analgesic. J. Am. Chem. Soc. 78:2342–2343 (May 1956).

INTRAOCULAR TENSION IN ASSOCIATION WITH SUCCINYLCHOLINE AND ENDOTRACHEAL INTUBATION: A PRELIMINARY REPORT

J. E. WYNANDS, M.D., and D. E. CROWELL, M.D.

IN 1953, Hoffman and Halzer reported the effects of ether anaesthesia and pentothal–nitrous oxide anaesthesia on intraocular tension. Since that time a good deal of interest has been shown in this field and in particular on the effect of the muscle relaxant, succinylcholine, on intraocular tension. Lincoff showed in animals that a step-wise dosage of succinylcholine produced proportional increases in intraocular pressure up to a maximum of 14 to 18 mm. Hg. Striate muscle tension was recorded and, as a control, succinylcholine was administered to produce 90 per cent paralysis of the gastrocnemius muscle. At this dosage succinylcholine produced the above-mentioned ocular results. Lincoff concluded that extraocular muscle shows a higher threshold to the effect of succinylcholine than does skeletal muscle and that the reaction to succinylcholine appears as a sustained contracture rather than as relaxation. They also concluded that the extraocular myoneural junction as opposed to that in skeletal muscle is not blocked by succinylcholine. In 1957, the above results were supported by the findings of Macri and Grimes and later in the same year by those of Dillon who carried out work on cat extraocular muscle *in vitro*.

On the basis of the above reports, we felt that the methods of investigation were incomplete in so far as it is reasonable to assume that, since the muscle-nerve ratio is 1:1 in extraocular muscle compared to 100:1 in skeletal muscle, a higher concentration of circulating succinylcholine will be required to achieve in extraocular muscle the same degree of relaxation produced in skeletal muscle. Therefore, when the gastrocnemius muscle is 80 per cent paralysed, the extraocular muscle should, theoretically, be paralysed to a far lesser degree.

As contraction of extraocular muscle increases intraocular tension, complete relaxation of extraocular muscle should result in lowered ocular tensions. This lowering of tension would take place if extraocular muscle responded to succinylcholine as does striate muscle: that is, there would be a period of fasiculation, which, when maximal, would be accompanied by a maximal rise in ocular tension that subsequently would fall to normal as the muscle became paralysed.

On the above hypothesis, an investigation was carried out using succinylcholine in association with light pentothal–nitrous oxide anaesthesia so that ocular tension variations might be studied via tonometry. Succinylcholine was employed under three different conditions and the resulting changes in intraocular tension were noted.

Series I. The effect of a single intubating dose of succinylcholine was measured, the time for return to normal being noted.

Series II. A 0.1 per cent succinylcholine drip was employed for four to five minutes until complete skeletal muscle paralysis was evident and the resultant changes in intraocular tension recorded.

Series III. The effect of a single intubating dose of succinylcholine immediately following complete skeletal muscle relaxation as achieved with the 0.1 per cent succinylcholine drip was observed.

Results

In Series I, 9 patients had an insignificant rise of 0 to 5 mm. Hg, 6 patients had a rise of 5 to 10 mm. Hg while 3 rose more than 10 mm. Hg, with one as high as 17. The 9 patients with a rise of 5 or more are significant. In Series II, 6 patients out of 36 had a significant rise of 5 to 10 mm. Hg, while 4 had a fall of 5 to 10 mm. Hg; 16 had an insignificant rise of 0 to 5 mm. Hg, while 7 had a fall of 0 to 5 mm. Hg. There were 3 patients without any change in tension. What is significant here is that 14 of the 36 patients had no change or a fall in ocular tension and that 16 had an insignificant rise. In Series III, 7 patients of 36 had a significant change in ocular tension, 3 having a rise and 4 a fall. What is most interesting is that 11 patients had no change in tension and 13 had an insignificant fall. Of the 3 patients with significant rises in ocular tension, it was felt that 2 had been inadequately depolarized with the 0.1 per cent succinylcholine drip as fasiculation of the extremities occurred with the large dose. Therefore, of the 36 patients in Series III, only one had a significant rise in intraocular tension.

TABLE I

Succinylcholine Effect on Ocular Tension

	Tension rise (mm. Hg)			No change	Tension fall (mm. Hg)		
Succinylcholine	0–5	5–10	10+		0–5	5–10	10+
Series I	9	6	3	—	1	—	—
Series II	16	6	—	3	7	4	—
Series III	5	2	1	11	13	2	2

TABLE II

Intraocular Tension in Association with Endotracheal Intubation

	Rise (mm. Hg)			
	0–5	5–10	10+	0
Without Topical	8	5	10	—
With Topical	6	2	0	3

In the course of studying the effect of succinylcholine on intraocular pressure, it was noted that endotracheal intubation immediately following the Series III study led to changes in ocular tension that frequently were far in excess of those produced by the succinylcholine. From Table II it may be seen that of 23 cases, 8 had an insignificant rise, while 15 had significant rises and of these 10 were highly significant, one being as high as 30 mm. Hg rise from normal. Further

investigation showed that if topical anaesthesia with 4 per cent Xylocaine was provided transtracheally 5 minutes prior to intubation the response to intubation was considerably altered. Of 11 cases, only 2 had a rise of 5 to 10 mm., 3 having no change, and 6 an insignificant rise. The rise in tension associated with intubation occurs rapidly and is not associated with a significant rise in venous pressure or arterial blood pressure. This increase in tension tends to last longer than that associated with succinylcholine and, when the tension has returned to normal, it may again rise with movement of the endotracheal tube. Carrying out the transtracheal injection is also associated with a rise in tension, but it is nearly always insignificant and always of very short duration.

Discussion

From the limited series presented, it appears that a single intubating dose of succinylcholine which wears off in four to five minutes results in a significant rise in ocular tension in about 50 per cent of individuals. This rise, however, is of short duration so that there is no contraindication to the use of succinylcholine for intubating purposes as the rise in intraocular pressure of 5 mm. Hg becomes significant only when the eye is open owing to the danger of vitreous being extruded. Where succinylcholine is used for intubation, the rise in ocular tension concomitant with muscle fasiculation and relaxation has long worn off before the surgeon is ready to open the eye.

From Series II, it can be seen that where depolarization of skeletal muscles is accomplished over four to five minutes with a 0.1 per cent succinylcholine drip so that fasiculation is not a feature of the depolarization, there is for all practical purposes no increase in intraocular tension. Where succinylcholine is desired for intubation and the surgeon is particularly anxious that there should be minimal changes in ocular tension, depolarization with a 0.1 per cent succinylcholine drip will provide the desired situation. If necessary, the succinylcholine drip may be maintained throughout the procedure as long as fasiculation is prevented.

Series III would seem to indicate that once a person is clinically depolarized the further administration of succinylcholine in large doses does not lead to an increase in intraocular tension and in some cases may actually produce a fall.

From Series II and III, which have gone further than previous studies in that, in one instance, complete depolarization of skeletal muscle has been obtained through slow depolarization, and, in the other instance, a large amount of succinylcholine has been given to such a depolarized patient without increase in ocular tension, we may conclude that one of two things happened.

1. The extraocular muscles do, in fact, behave like skeletal muscle with the exception that larger amounts of circulating succinylcholine are required to produce equivalent amounts of relaxation and that, as with skeletal muscle, if a large amount of succinylcholine is given quickly, fasiculations occur which cause a rise in intraocular pressure. If, however, fasiculation is prevented by slow depolarization, the rise in ocular tension is averted. The cases in which we had a significant rise in ocular tension were most probably due to too rapid depolarization as in some of these fasiculations were observed.

2. The other possibility is that perhaps the muscles do go into a sustained contracture, but when the succinylcholine is given slowly there is time for compensatory mechanisms within the eye as, for example, redistribution of aqueous; and that once the muscle is in full sustained contracture further administration of succinylcholine is ineffectual. A method of study is to be carried out which will determine the significant factor.

As regards the increase in ocular tension following endotracheal intubation, it appears to be of greater significance than the rise in tension associated with succinylcholine. It seems to be of reflex origin, being minimized by adequate topical anaesthesia.

Summary

There appears to be no reason why succinylcholine cannot be used for intubation where deemed advantageous by the anaesthetist, for the rise in intraocular pressure has been dissipated long before the surgeon is ready to open the eye. Where a rise may be undesirable, slow depolarization is indicated. Indeed, where a rise in ocular tension is feared, it would seem that that associated with intubation in the absence of adequate topical anaesthesia or moderately deep general anaesthesia may be of far greater and prolonged extent than that produced by succinylcholine. It remains a fact, however, that a single large injection of succinylcholine should never be administered when the eye is open during intraocular surgery.

Résumé

Chez 50 pour cent des individus, la succinylcholine à dose unique, en vue de pratiquer une intubation, entraîne une augmentation appréciable de la tension intra-oculaire. Toutefois, en deça de cinq minutes, la tension est revenue à ce qu'elle était, ce qui ne constitue pas une contrindication à l'emploi de la succinylcholine pour l'intubation des cas de chirurgie intra-oculaire puisque la tension est normale au moment où l'on ouvre l'œil. Si l'on emploie un goutte-à-goutte de succinylcholine à 0.1 pour cent pour dépolariser le malade de façon à éviter les fasciculations des extrémités, il n'existe plus, à toute fin pratique, d'augmentation de la tension oculaire. L'intubation endotrachéene s'accompagne également d'une élévation de tension intra-oculaire qui semble plus importante que celle qui suit l'administration de la succinylcholine. Cette augmentation de tension semble d'origine réflexe et elle serait diminuée par une anesthésie locale adéquate.

Acknowledgments

We wish to thank Dr. A. B. Noble, Director of Anaesthesia, Royal Victoria Hospital, Montreal, for his helpful guidance, and Dr. A. P. H. McLean for his assistance in carrying out this study.

REFERENCES

1. Colle, J., Duke-Elder, P. M., & Duke-Elder, W. S. Studies on the Intra-Ocular Pressure. J. Physiol. *71*: 1 (1931).

2. Drucker, A. P., Sadove, M. S., & Unna, K. R. Ophthalmic Studies of Curare and Curarelike Drugs in Man. Am. J. Ophth. *34*: 543 (1951).
3. Lincoff, H. A.; Ellis, C. H.; DeVoe, A. G.; DeBeer, E. J.; Impastato, D. J.; Berg, S.; Orkin, L.; & Magda, H. The Effect of Succinylcholine on Intraocular Pressure. Am. J. Ophth. *40*: 501 (1955).
4. Macri, F. J., & Grimes, P. A. The Effects of Succinylcholine on the Extraocular Striate Muscles and on the Intraocular Pressure. Am. J. Ophth. *44*(2): 221 (1957).
5. Lincoff, H. A., Breinin, G. M., & DeVoe, A. G. The Effect of Succinylcholine on the Extraocular Muscles. Am. J. Ophth. *43*: 440 (1957).
6. Dillon, J. B.; Sabawala, P.; Taylor, D. B.; & Gunter, R. Action of Succinylcholine on Extraocular Muscles and Intraocular Pressure. Anesthesiology *18*: 44 (1957).
7. Dillon, J. B.; Sabawala, P.; Taylor, D. B.; & Gunter, R. Depolarizing Neuromuscular Blocking Agents and Intraocular Pressure *in vivo*. Anesthesiology *18*: 439 (1957).
8. Sugar, H. S. Factors in Normal Tension, *in* The Glaucomas, chap. v, p. 55. 2nd ed.

OBSERVATIONS ON THE FOETUS DURING EXPERIMENTAL HYPOTHERMIA[1]

S. L. VANDEWATER, M.D., F.R.C.P.(C),[2] and
W. M. PAUL, M.D., F.R.C.S.(C)[3]

INDUCED HYPOTHERMIA has become, in many medical centres, an accepted adjunct in anaesthesia in the direct surgical approach in subarachnoid haemorrhage due to a ruptured berry aneurysm. This has been the practice in Toronto (1, 2) and, up to May 1959, 161 ruptured aneurysms had been operated upon, at varying times after the last haemorrhage, at the Toronto General Hospital. In this consecutive series, 91 patients were female, 26 of whom were less than 40 years of age. In 1956, Cannell and Botterell (3) reviewed a series of 12 patients with subarachnoid haemorrhage, complicating pregnancy, in six of which an aneurysm was demonstrated. Three of the six underwent common carotid ligation and survived, one had the middle cerebral aneurysm clipped successfully under induced hypotension (4), two died untreated, and one died following complications of arteriography. This report inspired the present work, which is an attempt to answer some of the questions on the immediate and delayed effects of hypothermia on the foetus. This may be considered an academic question, for our neurosurgical colleagues firmly believe in early operation and the use of hypothermia in the surgical treatment of subarachnoid haemorrhage.

METHOD

Observations were made on nine pregnant ewes, one to two weeks within term. The ewes, after being clipped and washed with a detergent to remove the skin oils, were given atropine 0.06 mg. intravenously, followed by thiopental sodium or pentabarbital sodium, and placed in the right lateral position. Because of the size and nature of the animals, and the presence of copious salivation, a tracheotomy was performed, and a cuffed tube inserted. A cannula was directed into the carotid artery, for mean maternal blood pressure recording and arterial sampling. The uterus was exteriorized, and the foetal circulation catheterized by the method described by Reynolds, Paul, and Huggett (5). A small incision was made in the uterus until the chorionic membrane was exposed. Branches of the umbilical cord were discovered, streaming between the amnion and the chorion, and a suitable vessel located. In this preparation a branch of the umbilical vein was catheterized and the uterine incision closed. It was thus possible to sample

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foetal blood with the foetus remaining *in utero*, and the membranes intact. In two experiments, the uterus was replaced in the abdominal cavity.

Maternal and foetal circulatory changes were noted, and oxygen saturation, carbon dioxide content, and pH estimations were made, using an Astrup apparatus.

Anaesthesia

In the first five experiments, the animals were induced with thiopental sodium (800–1,200 mg.), and anaesthesia maintained with a 50 per cent N_2O/O_2 mixture. The first two of this group were allowed to breathe spontaneously, with manual assistance, and chlorpromazine (100 mg.) was given in divided doses to combat shivering. Because of our inability to prevent respiratory acidosis, even with vigorous manual ventilation in these two experiments, the other three animals were curarized with d-tubocurare (18–30 mg.) and ventilated with a Jefferson positive-negative ventilator (+ 20, — 12 mm. Hg, 24–28 respirations per minute).

In the last four experiments, anaesthesia was induced with pentobarbital sodium (1,200–1,750 mg.), followed by curarization, and artificial positive-negative ventilation, using 100 per cent oxygen.

Hypothermia

Surface cooling was carried out using chopped ice, the application of ice on or near the exteriorized uterus being avoided. Ewe rectal and amniotic fluid temperatures were recorded with an electric thermocouple. The animals were cooled approximately 10° from the normal range of 39.0–39.5°C. "Crash" rewarming was partially carried out in seven of the nine animals using hot water.

Observations

The uterus was exteriorized for three to four hours, allowing exposure to a room temperature of about 22°C. This fact plus the taking of up to 50 cc. of blood (one-quarter of the foetal blood volume) contributed to the death of one foetus and terminal deterioration of others before crash rewarming was complete.

The maternal blood pressure was recorded as a mean pressure, and the foetal heart rate counted directly by palpation or auscultation. The blood gas estimations are presented with some reservations, because of the difficulty in arriving at a true figure when the blood has become cooled, and the animal has been pressure ventilated with oxygen. The pH estimations were adjusted to the temperature at the time the blood was taken (6). Barcroft (7), measuring foetal lamb oxygen saturation close to term, found values varying from 50 to 70 per cent. Reynolds and Paul (8), in similar preparations of foetal lambs at term, found the heart rate to vary from 140 to 200 per minute. In the experience of Paul (9) and Barn *et al.* (10), bradycardia does not usually occur until the foetal O_2 saturation falls to 10–15 per cent.

Experiment I

A moderate haemorrhage from the carotid artery occurred at 1½ hours, resulting in severe maternal hypotension. The maternal respiratory exchange even

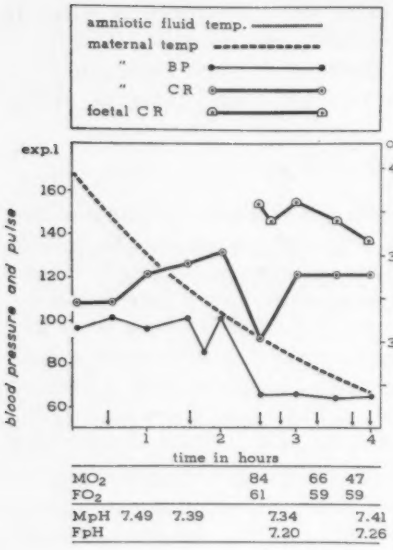


FIGURE 1

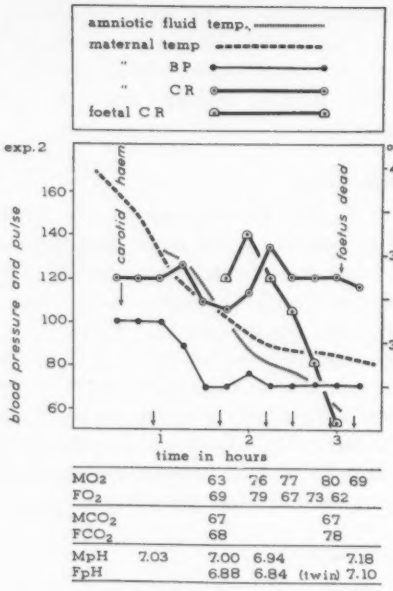


FIGURE 2

with vigorous manual assistance was inadequate, resulting in low maternal O_2 saturation. In spite of this state of hypotension and maternal hypoxia, the foetal heart rate and O_2 saturation remained within the normal range, up to the end of the experiment 1½ hours later. This was during the hypothermic period, and no rewarming was carried out. The foetal pH values paralleled those of the ewe, but were lower. (See Fig. 1.)

Experiment II

Gross haemorrhage occurred from a dislodged carotid cannula, resulting in maternal hypotension. Maternal respiratory exchange was inadequate (with manual assistance) leading to hypoxia and respiratory acidosis. In spite of this, the foetal O_2 saturation remained high, but there was a steady decline in cardiac rate and pH, and a rise in CO_2 content. It would appear, that in this experiment the bradycardia and eventual death of the foetus were not due to foetal anoxia, but were related to maternal acidosis. (See Fig. 2.)

Experiment III

Controlled respirations with a Jefferson ventilator were carried out in this and the following experiments. The blood samples were unsuitable for gas analysis. The maternal and foetal pH readings showed a fall followed by a rise, after an increase in the respiratory pressures, and aspiration of secretions through the tracheotomy tube. The fall in foetal cardiac rate followed the pH changes which might suggest the effect of maternal acidosis. (See Fig. 3.)

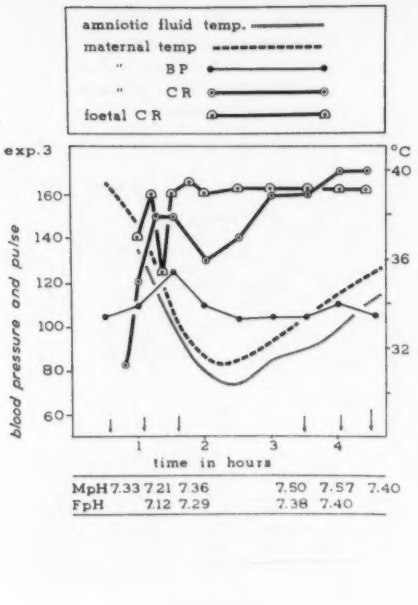


FIGURE 3

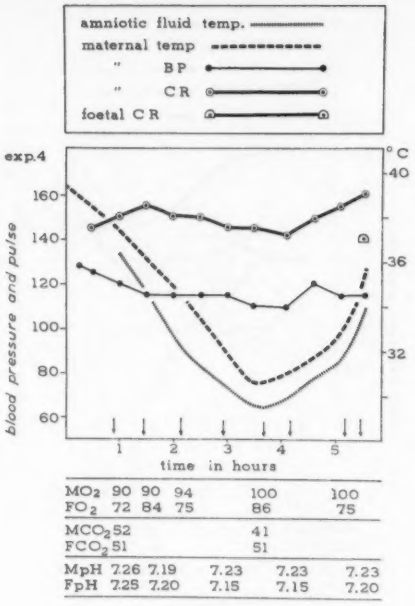


FIGURE 4

Experiment IV

The foetal pulse was not available as the uterus has been replaced in the abdomen. All figures were within normal limits except for the rather low pH. The foetal O_2 saturation figures were unusually high. (See Fig. 4.)

Experiment V

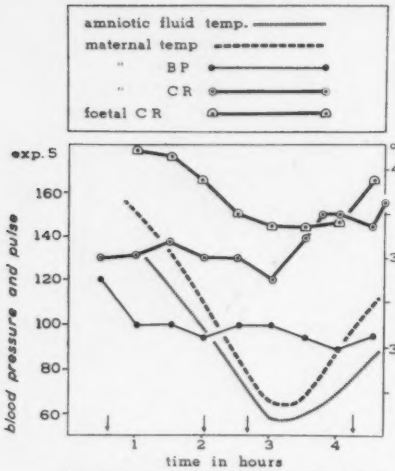
All figures were within normal limits, except the pH was again rather low. The foetal O_2 saturation figures were normal, with a normal foetal pulse rate throughout the four hours of exposure. (See Fig. 5.)

Experiment VI

Pentobarbital sodium, curare, controlled respirations and 100 per cent O_2 were used in this and in the following three experiments. The uterus was replaced in the abdomen. Rapid rewarming with hot water (50°C.) resulted in a slight fall in maternal blood pressure, probably as a result of vasodilatation. The foetal O_2 remained within normal limits, although the CO_2 content rose and pH fell. The foetus survived the time of the experiment. (See Fig. 6.)

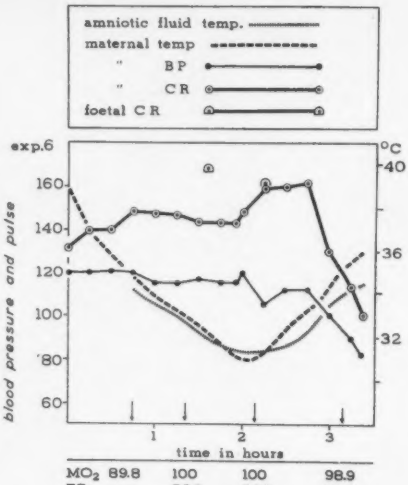
Experiment VII

There was a severe fall in maternal blood pressure and pulse rate with the onset of rewarming. The blood pressure was restored with vasoxamine, 10 mg., intravenously. The ewe was difficult to oxygenate, even with high pressures, in



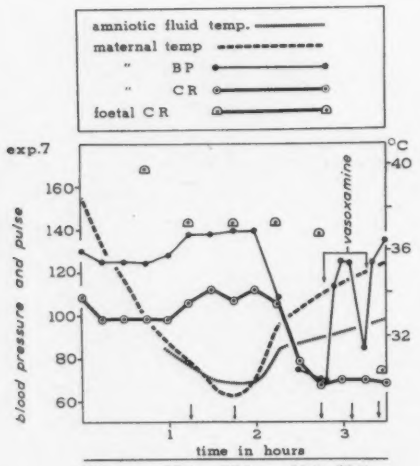
MO_2	92	98	93	88
FO_2	71	78	77	75
MCO_2	45		40	
FCO_2	47		52	
MpH	7.39		7.27	7.21
FpH	7.18		7.31	7.18

FIGURE 5



MO_2	89.8	100	100	98.9
FO_2		56.2	52.7	66.0
MCO_2	54.0	57.1	51.8	45.9
FCO_2	55.8	62.1	68.8	71.9
MpH	7.312	7.294	7.330	7.262
FpH	7.132	7.224	7.237	7.072

FIGURE 6



MO_2	67.4	78.9	68.7	68.0	
FO_2	56.1	74.6	51.6	23.8	
MCO_2					
FCO_2					
MpH	7.116	7.130	7.064	7.069	7.057
FpH	7.106	7.130	7.059	7.039	6.952

FIGURE 7

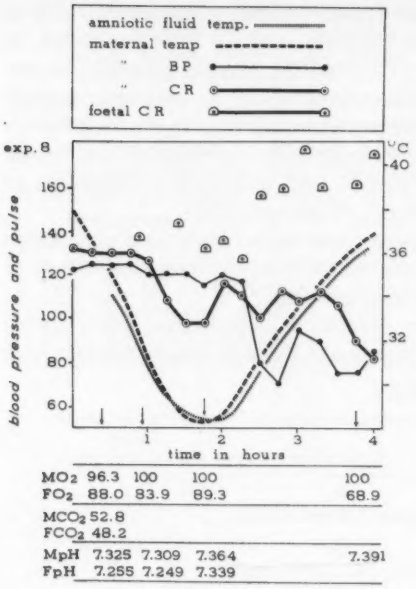


FIGURE 8

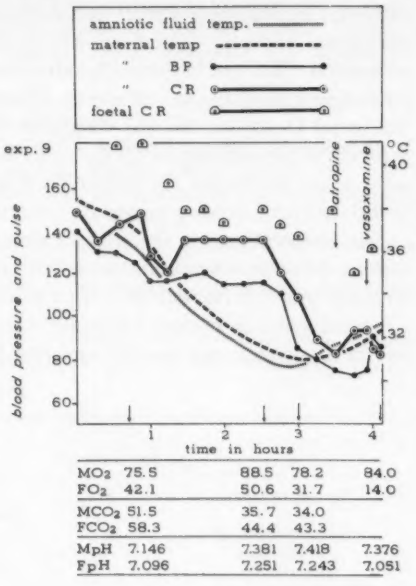


FIGURE 9

part due to copious secretions requiring frequent aspiration. The foetal O_2 saturation was surprisingly maintained, until near the end of the experiment. Both the maternal and the foetal pH values were low. Measurement of CO_2 content was not done. (See Fig. 7.)

Experiment VIII

There was a fall in maternal blood pressure with rewarming. All figures were normal throughout the experiment, with the foetus quite active and alive at the end. (See Fig. 8.)

Experiment IX

Again, there was a severe fall in maternal blood pressure and pulse rate with rewarming. Both maternal and foetal O_2 saturations were low, with the foetus deteriorating towards the end of the experiment. The CO_2 determinations were normal. The pH values increased during cooling, but the foetal pH was quite low at the final reading. The foetus survived the experiment, but was in poor condition. (See Fig. 9.)

DISCUSSION

Maternal respiratory acidosis and hypoxia could only be prevented by using relatively high inflationary pressures with an automatic respirator in a curarized pregnant sheep lying on its side. Hypotension from haemorrhage, with adequate oxygenation did not adversely affect foetal heart rate or O_2 saturation. However

maternal hypotension which occurred with rapid rewarming presumably due to peripheral vascular dilatation, was usually accompanied by a falling foetal O_2 saturation. Changes in foetal O_2 saturation, CO_2 content, and pH generally accompanied similar changes in the ewe. When measurements in the ewe were normal, the foetal O_2 saturation was well above the recognized normal level. Respiratory acidosis in the ewe appeared to cause severe bradycardia and death of one foetus, even when the foetal O_2 saturation was normal, and there was a suggestion of foetal distress (slowing heart rate) in a second (Experiment III).

The observations on the sheep foetus were acute experiments, and no attempt was made to save the animals. A brief study on the possible long-term effects of hypothermia was carried out with a series of guinea pigs.

Foetal survival studies following single episodes of hypothermia were carried out in the guinea pig in mid- and late pregnancy (Table I). Body temperatures

TABLE I

	No. animals	Total litter	Litter survival (1 wk.)
No hypothermia	16	61 (average 3.8 per animal)	57
Hypothermia at 55–65 days gestation	6	25 (average 4.1 per animal)	24
Hypothermia at 30–40 days gestation	8	29 (average 3.6 per animal)	27

were lowered 10–12°C. by immersion of the unanaesthetized animals in ice water. A comparison of litter size and survival was made between this group and control animals in the same colony. It is noted that single episodes of hpothermia of this degree did not alter size or survival in these animals.

Conclusions

No conclusions can be made from these limited experiments. However, we might infer that foetal distress occurred during hypothermia with the occurrence of maternal respiratory acidosis and hypoxia, and with gross maternal hypotension due to peripheral vascular dilatation from rapid surface rewarming. It would appear that well-conducted anaesthesia and hypothermia may be a safe procedure for the foetus.

REFERENCES

1. Botterell, E. H.; Lougheed, W. M.; Scott, J. E.; & Vandewater, S. L. Hypothermia, and Interruption of Carotid or Carotid and Vertebral Circulation, in the Surgical Management of Intracranial Aneurysm. J. Neurosurg. *13*: 1 (1956).
2. Botterell, E. H.; Lougheed, W. M.; Morley, T. P.; & Vandewater, S. L. Hypothermia in the Surgical Treatment of Ruptured Intracranial Aneurysms. J. Neurosurg. *15*: 4 (1958).
3. Cannell, D. E., & Botterell, E. H. Subarachnoid Haemorrhage and Pregnancy. Am. J. Obst. & Gynec. *72*: 844 (1956).

4. VANDEWATER, S. L. The Use of Hypotension and Hypothermia in Subarachnoid Haemorrhage. Symposium on "Pathogenesis and Treatment of Cerebral Vascular Disease," vol. 5, Houston Neurological Society, Houston, Texas. Springfield, Ill.: Charles C. Thomas, in publication (1959).
5. REYNOLDS, S. R. M., PAUL, W. M., & HUGGETT, S. St. G. Physiological Study of the Monkey Foetus *in utero*. Bull. Johns Hopkins Hosp. *95*: 256 (Nov. 1954).
6. CRAIG, F. A.; LANGE, K.; OBERMAN, J.; & CARSON, S. A Simple Accurate Method of Blood pH Determination for Clinical Use. Arch. Biochem. Biophys. *38*: 257 (1952).
7. BARCROFT, Sir JOSEPH. Researches in Prenatal Life, vol. 1. London: Blackwell Scientific Publications (1946).
8. REYNOLDS, S. R. M., & PAUL, W. M. Relation of Bradycardia and Blood Pressure of the Foetal Lamb-*in-utero* to Mild and Severe Hypoxia. Am. J. Physiol. *193*: 249 (1958).
9. PAUL, W. M. A Mechanism of Bradycardia in the Foetal Lamb-*in-utero*. Bull Johns Hopkins Hosp. *99*: 357 (Dec. 1956).
10. BORN, G. V. R., DAWES, G. S., & MOTT, J. C. Oxygen Lack and Autonomic Nervous Control of the Foetal Circulation in the Lamb. J. Physiol. *134*: 149 (Oct. 1956).

SECOND THOUGHTS ON HALOTHANE IN NEUROSURGERY

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It is now more than two years since the representatives of Canadian universities reported before the Society their initial studies with halothane, and in the interim the pharmacology and clinical use of halothane have been extensively investigated. Though the cardiovascular effects of halothane and of chloroform are strikingly similar, there is as yet no concrete evidence that halothane is a potential liver poison when administered with the aid of modern anaesthetic techniques (whose superiority to those available when chloroform was first employed is obvious). It can be assumed, therefore, that halothane merits a secure place in anaesthesia although it is open to question whether future progress in anaesthesia depends upon the use of the volatile anaesthetics.

We have found halothane to be a valuable agent in neurosurgical anaesthesia, and report here some general impressions derived from our experience with this agent.

Radiological Investigations

One indication for the use of halothane is the arteriogram or other procedure requiring light anaesthesia, where an explosive hazard exists. Following intubation by the thiopental-relaxant technique, light anaesthesia with 0.5–1.0 per cent halothane in 50 per cent nitrous oxide–oxygen has replaced former methods involving the use of trichlorethylene with or without intermittent thiopental injections. In a series of 179 patients (ages 6–66 years) anaesthetized in this way with halothane, systolic blood pressure levels below 90 mm. Hg occurred at some time during the administration in 32 patients, an incidence of 18 per cent. This degree of hypotension may, therefore, be expected in one out of every five or six patients adequately anaesthetized with nitrous oxide–oxygen–halothane for minor or superficial surgical procedures. In this series, blood pressure frequently returned to levels above 90 mm. Hg, either spontaneously or by a reduction in the inspired halothane concentration, but vasopressor drugs, usually methoxamine, were given in 13 of the 32 patients showing levels below 90 mm. Hg: that is, falls in blood pressure during halothane administration were considered to require treatment in only 7 per cent of the patients in this series. The procedures have been otherwise uneventful, without troublesome cardiac arrhythmias or falls in blood pressure associated with dye injection.

Surgery of the Vertebral Column and Spinal Cord

Halothane has played an important part in the major change in anaesthetic technique which has resulted from the use of controlled respiration in neuro-

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surgery in this unit. Thus, positive-negative phase mechanical ventilators have been used in conjunction with nitrous oxide–oxygen–halothane anaesthesia in more than 150 major neurosurgical procedures, most of which involved an extensive and sometimes prolonged removal of single or multiple lumbar intervertebral disc protrusions. In this field spinal anaesthesia was previously found to be especially successful in minimizing epidural venous bleeding, producing distinctly better operating conditions than general anaesthesia with spontaneous respiration. It is our opinion, however, that mechanical ventilators have revolutionized general anaesthetic techniques, giving results which equal or surpass conditions thought to be attainable only by spinal (or epidural) blockade.

Halothane may be safely employed with mechanical respirators provided that vapour concentration is controlled independently of tidal volume. We have continued to employ the Marrett apparatus, with gas flows up to 10 L. per minute and with the "trichlorethylene" bottle containing 2 oz. of halothane. The concentration of halothane feeding the respirator does not exceed 1.5 per cent, therefore, and below 1 per cent is usually sufficient. The Bennett "assister" has been the preferred type, used only as a controlled respiration unit, with pressures of about +15 and −5 cm. H_2O.

After intubation with the thiopental-relaxant technique (or under thiopental anaesthesia induced after transtracheal injection of 4 per cent Xylocaine), apnoea can frequently be maintained with nitrous oxide–oxygen halothane alone. More usually, injections of tubarine maintain relaxation, and we have not found the combination of tubarine and halothane to present any special problem; although a variable, usually small, fall in systolic pressure occurs within a few minutes of injecting tubarine (not only during halothane anaesthesia), in our experience an adequate pressure level is re-established after a few minutes without the need for vasopressors.

With this technique, excellent conditions are obtained for the surgery of the spinal canal. Venous drainage of the epidural veins appears excellent, the spinal dura visibly expanding and collapsing during the positive and negative respirator phases. It is our view that when optimal ventilation is achieved by means of positive-negative pressure ventilation, management of the circulation is simplified. By judicial increases in halothane concentration, controlled degrees of arterial hypotension can provide a dry surgical field. It should be emphasized that patients with spinal fractures are highly sensitive to the hypotensive action of halothane, and accurate blood pressure measurement is essential for the safe management of such patients, who behave similarly during either spontaneous or controlled respiration.

Intracranial Procedures

For many years, and in spite of theoretical objections, nitrous oxide–oxygen–ether has been the mainstay in the anaesthetic management of all varieties of neurosurgical procedures in this unit. Halothane is now a valuable adjunct, and under certain circumstances has replaced ether, allowing a more rapid recovery from prolonged neurosurgical procedures. Unfortunately, the use of halothane is restricted by surgical infiltration of the operation site with local analgesic solution

containing adrenalin, which introduces an unpredictable hazard, necessitating the temporary use of other agents (thiopental, Demerol, or ether) for periods of at least ten minutes before and 30 minutes after injection of analgesic solution.

The reduction in haemorrhage obtained by the use of positive-negative pressure ventilation is less obvious in intracranial surgery than in spinal procedures. Clearly, when intracranial tension is increased by a space-occupying lesion, mechanical ventilators will be relatively ineffective in promoting a fall in cerebrospinal fluid and intracranial venous pressures. On the other hand, sudden increases in intracranial tension, secondary to coughing or straining, are absent, so that the use of the respirator technique when properly managed is a valuable preventive measure against the poor operating conditions sometimes seen during general anaesthesia with spontaneous respiration.

As an aid in the intracranial exposure of vascular aneurysms, pituitary tumours, and acoustic nerve tumours, the halothane/respirator technique is excellent; in combination with lumbar drainage, or with intravenous injection of urea solutions, maximum brain shrinkage is obtainable. Here, also, the hypotensive properties of halothane are advantageous. When hypertension accompanies brain swelling, secondary to haemorrhage near the brain stem, halothane has been given in stronger concentrations to reduce blood pressure and minimize further brain swelling.

Résumé

En anesthésie neurochirurgicale, nous avons trouvé que l'halothane est un agent de valeur. L'emploi de l'halothane trouve une indication dans les cas d'artériogrammes et dans d'autres circonstances où une anesthésie légère est requise et où il existe un risque d'explosion. On peut s'attendre à une hypotension systolique au-dessous de 90 mm. Hg chez un sur cinq ou six malades adéquatement anesthésiés avec protoxide d'azote–oxygène–halothane pour des interventions chirurgicales mineures ou superficielles.

Nous avons employé des ventilateurs à phase positive-négative au cours de l'anesthésie avec protoxide d'azote–oxygène–halothane pour au-delà de 150 opérations majeures neurochirurgicales dont la plupart consistaient en l'ablation extensive et quelquefois prolongée de disques intervertébraux lombaires herniés. Nous sommes d'avis que, dans ce domaine, les ventilateurs mécaniques ont révolutionné les techniques d'anesthésie générale en permettant d'obtenir des résultats égaux et même surpassant ce que nous croyions pouvoir obtenir seulement avec la rachianesthésie ou le blocage épidural.

Avec les respirateurs mécaniques, il est possible d'employer l'halothane pourvu que, en dehors de l'air courant, la concentration des vapeurs soit contrôlée.

Pour l'anesthésie au cours des opérations intracrâniennes, l'halothane est une précieuse acquisition qui, en certaines circonstances, a remplacé l'éther en permettant un réveil plus précoce à la suite d'opérations neurochirurgicales prolongées. Mentionnons, toutefois, que l'emploi de l'halothane n'est pas compatible avec l'infiltration dans le site opératoire d'un anesthésique local contenant de l'adrénaline, ce qui pourrait apporter un risque imprévisible. La diminution de l'hémorragie obtenue par l'usage de la ventilation à pression positive et négative

est moins évidente au cours de la chirurgie intracrânienne qu'au cours des opérations sur la colonne vertébrale.

Les vertus hypotensive de l'halothane présentent un avantage en aidant à visualiser dans le crâne les anévrysmes vasculaires, les tumeurs de la pituitaire et les tumeurs du nerf acoustique. Quand l'hypertension s'accompagne d'un gonflement du cerveau, nous avons donné des concentrations plus élevées d'halothane dans le but de réduire la tension sanguine et de diminuer le gonflement cérébral.

REFERENCE

BRINDLE, G. F., GILBERT, R. G. B., & MILLAR, R. A. The Use of Fluothane in Anaesthesia for Neurosurgery: A Preliminary Report. Canad. Anaesth. Soc. J. *4*: 265 (July 1957).

BACTERIOLOGICAL CONTROL IN A BLOOD BANK: A REPORT

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AMONG THE AUTHORS who have studied bacterial contaminants of blood, Braude (1) reports contamination by gram-positive and gram-negative microbes. The former comprise pathogenic staphylococci and diphtheroids which do not cause serious reactions in man. The latter are much more dangerous and are known to be responsible for severe shocks on account of their endotoxins (2, 3, 4). As stated by Braude, Corey, and Siemienski (5) these gram-negative germs can be divided into two groups according to their physiology:

1. A first group comprises saprophytic species belonging to the genera *Pseudomonas* and *Achromobacter*. These bacilli are cryophillic: their optimum temperature being between 4° and 8° C.; when incubated at 37° C., the growth is very poor and slow to develop.

2. A second group contains microbes of the coli-aerogenes group: *E. coli, E. freundii, E. intermedium*. Pittman (6) reports other gram-positive contaminants such as: streptococci, sarcina, and *B. cereus*. He also cites blood contamination by *Paracolon intermedium, Paracolon aerogenoides, Faecalis alcaligenes*, and other germs capable of utilizing citrate as the sole source of carbon. In eighteen fatal transfusions, he has found eight strains of the genus *Pseudomonas*. However, *Pseudomonas aeruginosa* was not involved in these accidents.

According to Braude (1), the rate of contamination in blood banks is between 2 and 3 per cent. Reactions are rare and occur only when blood is heavily contaminated. In England, during the war, 1939 to 1945, blood contamination in blood banks was about 5 per cent according to Whilly (7), while in New York, during the same period, Heath and Angrem (8) reported an 8.5 per cent contamination calculated on a quantity of 6,151 L. blood. Walter, Kundsin, and Button (9) report 47 contaminations from 4,497 specimens (1.04 per cent) in which were found *Staph. aureus*, streptococci, a flavobacterium, yeasts, aspergilli, and penicillium.

The reason why slight contamination is not very dangerous is that fresh blood has antibacterial properties apparently related to the presence of antibodies and complement as pointed out by Geller, Chandler and Janetz (10), also of properdin according to Wardlay and Pillemer (11). These properties seem to be enhanced by gram-negative microbes following a short period of incubation at 37° C., before the storage of the blood in the bank; this activity is not so evident for cryophillic bacteria which later multiply in the refrigerator. Low temperatures are believed to inhibit these antibacterial factors. This present paper deals with the search for bacterial contaminants in a normally operated blood bank. Its aim was to control the sterility of the blood and to compare methods and results with the data from the above-mentionned authors.

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Material and Technique

The samples were taken from the blood bank of the Hôtel-Dieu Hospital in Quebec City during October 1958, the blood having been kept in the refrigerator at 6° to 8° C. for periods varying from two to 31 days. By preference, the oldest specimens were chosen so that even the slowest growing organisms such as spores, fungi, or cryophillic bacteria might be detected. Blood specimens were taken from the containers (rubber caps well cleaned with 70° alcohol) in a most aseptic manner by the anaesthetist and the bacteriologist assisted by a nurse, all three wearing masks. 2.5 ml. were withdrawn and inoculated immediately into a test tube containing 7.5 ml. thioglycollate liquid medium.[3] (Thioglycollate medium has long been recommended for sterility tests; it may be used alone, with methylene blue or even better still with resazurin as an rh indicator, Pittman (12)). The last drop of the blood in the syringe was used for a direct examination. In this way, 58 specimens were collected; of these, 30 were taken from glass containers,[4] the remaining from plastic bags.[5] All specimens were incubated for one month: 15 days at 37° C. and 15 days at room temperature (20°).

Following this incubation period, a second direct examination was made for all tubes and a gram staining was done whenever necessary. May we stress the necessity of making direct examination, because apparently one cannot rely on the clarity of the culture media to conclude that they are sterile; as a matter of fact, Wichelhausen, Clark, Griffing, and Robinson (13) have shown that a solution of human serum-albumin diluted to 25 per cent may be quite contaminated without showing any turbidity. These authors have thus isolated from

TABLE I

Number of cases	Storage times (days) at 6 to 8°C.	Results obtained after 15 days at 37°C. and 15 days at 20°C.
1	31	
1	30	
3	23	
2	21	
1	20	Both direct examinations and cultures were negative.
4	19	
1	17	
3	16	
3	15	
3	13	
2	11	
3	10	
5	9	
12	8	
2	7	Three specimens gave a culture of *Faecalis alcaligenes.*
3	6	
4	5	Both direct examinations and cultures were negative.
5	2	
58		

[3]No 01-140, Baltimore Biological Laboratory Inc.
[4]Transfuso-vac., F. 83, Baxter Laboratories of Canada, Acton, Ont.
[5]Pliapak A.D.C. Solution, Abbott Laboratories, Montreal.

clear sera the following organisms: *C. albicans*, *Proteus vulgaris*, *P.aeruginosa*, and *Streptococcus faecalis*.

Table I summarizes the procedures followed and also the results.

Results and Discussion

Only three specimens of blood from glass containers gave positive cultures (*Faecalis alcaligenes*) after 25 days of incubation. It is most probable that these specimens were contaminated in the laboratory, as: (i) the cultures were examined macroscopically every day, and turbidity appeared only on the twenty-fifth day, after 15 days of incubation at 37° C., and 10 days at 20° C.; (ii) the plasma haemoglobin content of the three specimens of blood was very low; (iii) the patients who received the blood presented no reaction whatever.

These contaminations have undoubtedly occurred through the cotten plugs of the culture tubes. In order to prevent such casualties, it may be advisable to seal the tubes or to use rubber caps. According to Marfarlane, Mainwaring, Maesween, and Parish (14), it is most difficult to avoid contamination when cultures have been inoculated for a long period of time, and in order to overcome this difficulty they described a method for sealing the tubes.

As noted previously, only massive contaminations cause serious post-transfusion accidents. It may be of interest to recall briefly that reactions occur thirty minutes or more after the transfusions. The symptoms are chills, temperature, muscular algia, hypotension, and shock which may be fatal (15–17). As stated by Braude (1), such accidents can be prevented by taking the following precautions: (i) a direct examination of the blood immediately before the transfusion; (ii) a routine use of 20 mg. of tetracycline per litre of blood. Braude recommends these precautions when storage conditions are poor, or when difficult manipulations are inevitable as in wartime or during a civil catastrophe.

In order to prevent an eventual multiplication of microbes, the time lapse between the collection of the blood and the transfusion should be as short as possible. According to Braude (1), the bacterial multiplication may reach important proportions in a week or two, in such a manner as to give concentrations of 10^9 to 10^{11} germs per millilitre.

We believe that whenever procedures of collecting blood have been long and difficult, the laboratory should proceed to immediate cultures and that such blood should be used within a week. A direct examination and a gram staining made immediately before the transfusion are advised. Stevens, Legg, Henry, Dille, Kerby, and Finch (4) recommend a staining just before transfusion. According to Walter *et al.* (9), a sterility test should be made before a transfusion, whenever the blood has been refrigerated for more than 96 hours. These same authors also describe a new technique for the detection of blood contaminants by the use of plastic equipment which seems to lessen the risks of contamination, in comparison to glass containers.

Conclusion

After 25 days of incubation three blood bank specimens out of 58 were con-

taminated with *Faecalis alcaligenes*. These contaminations occurred in the laboratory, as the other 55 samples remained sterile even after one month of incubation. This was made evident by a gram stain of the cultures.

In a well-organized blood bank, contamination can be avoided, at least in blood specimens used for transfusions, by having this service conducted under the supervision of an anesthetist working in close co-operation with the laboratory staff.

Summary

Fifty-eight specimens of blood from a blood bank were inoculated in fluid thioglycollate, and incubated 15 days at 37° C., and 15 days at 20° C., for sterility control; only three yielded a culture of *Faecalis alcaligenes* on the twenty-fifth day of the experiment, and it is assumed that this was caused by a contamination in the laboratory.

A general review of the subject is given, along with a discussion of the prophylaxis of blood bank contamination.

Résumé

Cinquante-huit spécimens venant d'une banque de sang ont été ensemencés dans du thioglycollate liquide et incubés durant 15 jours à 37° C., et durant 15 autres jours à 20° C., pour contrôle bactériologique. Trois échantillons ont donné une culture de *Faecalis alcaligenes* au vingtième jour de l'expérience; selon toute évidence, il s'agissait d'une contamination survenue au laboratoire.

Une revue générale de la question est éxposée, ainsi que les moyens à prendre pour prévenir la contamination du sang des banques.

REFERENCES

1. Braude. A. J. Transfusion Reactions from Contaminated Blood. New Eng. J. Med. 258:1289 (1958).
2. Braude, A. J.; Williams, D.; Siemienski, J.; & Murphy, R. Shock-like State due to Transfusion of Blood Contaminated with Gram-negative Bacilli: Successful Treatment with Antibiotics and Arterenol. Arch. Int. Med. 39:1228 (1953).
3. Borden, C. W., & Hall, W. H. Fatal Transfusion Reactions from Massive Bacterial Contamination of Blood. New Eng. J. Med. 245:760 (1951).
4. Stevens, A. R.; Legg, J. S.; Henry, B. S.; Dille, J. M.; Kerby, W. N. M.; & Finch, C. A. Fatal Transfusion Reactions from Contamination of Stored Blood by Cold Growing Bacteria. Ann. Int. Med. 39:1225 (1953).
5. Braude, A. J., Corey, P. J., & Siemienski, J. Studies of Bacterial Transfusion Reactions from Refrigerated Blood: Properties of Cold Growing Bacteria. J. Clin. Invest. 39:311 (1955).
6. Pittman, M. Study of Bacteria Implicated in Transfusion Reactions and of Bacteria Isolated from Blood Products. J. Lab. & Clin. Med. 42:273 (1953).
7. Whilly, L. Blood Transfusion, ed. G. Keynes. Bristol: John Wright & Sons Ltd. (1940).
8. Heath, F. K., & Amgren, J. Preservation of Human Plasma: Report of Studies with Sulfonamide Compounds. J.A.M.A. 118:1034 (1942).
9. Walter, C. W., Kundsin, R. B., & Button, L. N. New Technic for Detection of Bacterial Contamination in a Blood Bank using Plastic Equipment. New Eng. J. Med. 257 (7): 364–369, (1957).

10. GELLER, P., CHANDLER, A. B., & JANETZ, E. Experimental Studies on Bacterial Contaminations of Bank Blood. J. Lab. & Clin. M. *49*(4):573 (1957).
11. WARDLAW, A., & PILLEMER L. The Properdin System and Imunity. J. Exper. Med. *103*:553 (1956).
12. PITTMAN, M. A Study of Fluid Thioglycollate Medium for the Sterility Test. J. Bact. *51*:19–30 (1946).
13. WICHELHAUSEN, R. H.; CLARK, H. W.; GRIFFING, V. F.; & ROBINSON, L. B. The Concealment of Heavy Bacterial Contamination in 25 per cent Human Serum Albumin: Its Mechanism and Clinical Significance. J. Lab. & Clin. Med. *51*:277 (1958).
14. MACFARLANE, R. C.; MAINWARING, J. C.; MAESWEEN, J. C.; & PARISH, H. J. Technique for the Filtration of Human Plasma and Serum for Transfusion. Brit. Med. J. *1*:377 (1942).
15. BRAUDE, A. J.; SIEMIENSKI, J.; WILLIAMS, D.; & SANDFORD, J. P. Overwhelming Bacteriemic Shock Produced by Gram-positive Bacilli: Report of Four Cases with One Recovery. Univ. Michigan M. Bull. *19*:23 (1953).
16. WEIL, M. H., & SPINK, W. W. Shock Syndrome Associated with Bacteremia due to Gram-negative Bacilli. Arch. Int. Med. *101*:184 (1958).
17. HALL, H. W., & GOLD, D. Shock Associated with Bacteremia: Review of Thirty-five Cases. Arch. Int. Med. *96*:403 (1955).

THE PRESERVATION OF HUMAN BLOOD IN GLASS AND PLASTIC CONTAINERS: AN *IN VITRO* EVALUATION

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BECAUSE OF conflicting reports in the literature concerning the effects of plastic containers on red blood cells (1–7), there has been and still is some hesitation in substituting plastic for glass containers for blood storage. As evidence of this uncertainty, there is the known fact that many of the blood banks are still using both types of container indiscriminately. In order to throw light upon the problem, our present investigation was undertaken with the practical objective of finding out which is less detrimental to red blood cells. However, it must be

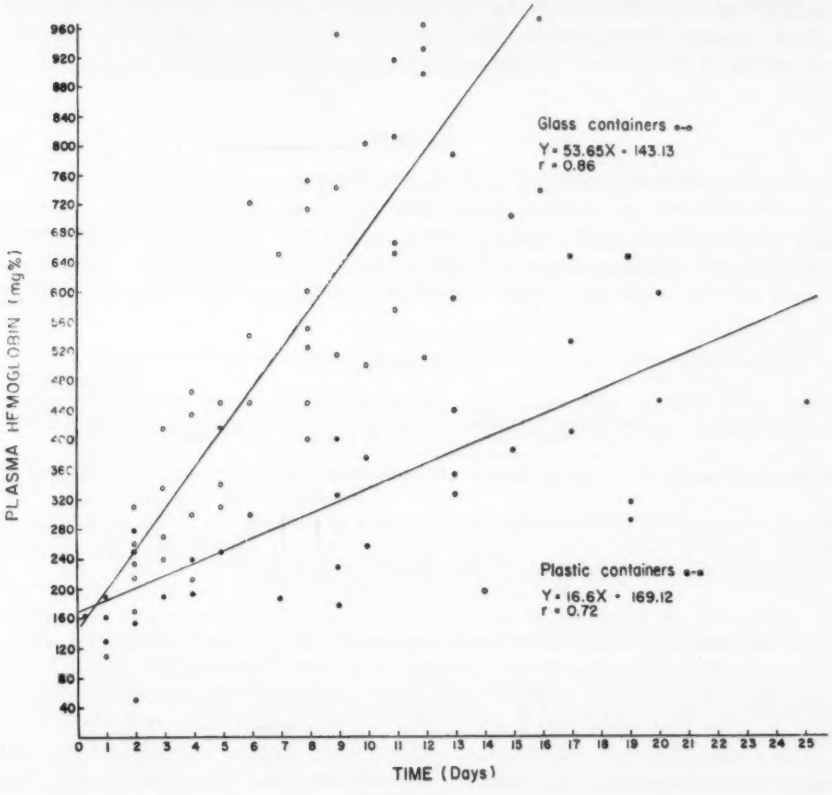

FIGURE 1

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emphasized that this study was intended to compare only the effectiveness of glass and plastic containers in the preservation of blood, not their individual differences.

Experiments

During a one-month period, blood was withdrawn from one group of 31 human donors and stored in glass containers[3] and from another group of 48 and stored into plastic containers.[4] Donors were male and female healthy persons. The blood specimens were then kept in a refrigerator at a temperature ranging from 6 to 8° C. and taken out only at the end of the collection period for the sampling procedure.

At the time of sampling, a 10 ml. blood sample was drawn from each container by means of a syringe and a 16-gauge needle, then gently poured into a Lusteroid type of centrifuge tube. The centrifugation was carried on in the cold (0–4° C.) at 2,500 r.p.m. for fifteen minutes. The plasma was decanted and kept for haemoglobin content determination. The plasma haemoglobin content was estimated according to the method recommended by Storck and Ardry (8).

Results

The haemoglobin content of each plasma sample was expressed in milligrams per 100 millilitres and plotted against time of storage (Fig. 1). Circles represent the results obtained with glass (vacuum bottle) containers and dots the results obtained with plastic containers. In both sets of results, the curve of best fit was found and the equation of their respective regression curves indicated thereby.

TABLE I

	Correlation coefficient $r \pm \sigma r$	Equation of regression curves $Y = mX + k$	$\pm \sigma m$*	Standard error of the sample in absolute value Sy†
Glass containers	8.86 ± 0.075	$Y = 53.65X + 143.13$	4.59 (1) 2.74 (2)	125.97
Plastic containers	0.72 ± 0.123	$Y = 16.6\ X + 169.12$		106.26

σm = Standard error of angular coefficient. The t value between σm_1 and σ_2 is 6.9.
†This standard error of the sample is given in the same unit as Y.

As indicated in Table I, the correlation coefficient ($r \pm \sigma r$) of the two series of results was highly significant. Actually, an r value equal to 0.86 ± 0.075 was calculated from results obtained with glass containers, whereas an r value of 0.72 ± 0.123 was calculated from results obtained with plastic containers.

The t value between the standard error of each angular coefficient (σm_1 and σm_2), derived from the equations of regression curves, was equal to 6.9. The difference therefore is highly significant.

[3]Transfuso-vac., F.83, Baxter Laboratories of Canada, Acton, Ont.
[4]Pliapak A.D.C. Solution, Abbott Laboratories, Montreal, P.Q.

Discussion and Conclusion

It is noteworthy that, in spite of our scattered results, in both cases the r values are very good. The correlation between the factors "time of storage" and "haemolysis of stored blood cells" is real and directly proportional. According to Strumia, Colwell, and Dugan (9), this red blood cell damage might be attributable to toxic substances leached from the material, glass or plastic, of which the containers are made.

Of more interest is the t value. It testifies strongly to the significance of our results and indicates that the rate of haemolysis is far more rapid when blood is stored in glass containers than in plastic containers.

Summary

Plasma haemoglobin content was determined in samples of blood withdrawn from glass and plastic containers where it had been stored for different periods of time. Our results indicate that the rate of haemolysis is far more rapid when the blood is stored in glass than in plastic containers.

Résumé

L'hémoglobine plasmatique a été mesuré dans des échantillons de sang d'âge différent venant de récipients en verre et en plastique.

Nos résultats indiquent que la vitesse d'hémolyse sanguine est beaucoup plus rapide dans le sang conservé dans des récipients en verre que dans celui conservé dans des récipients en plastique.

REFERENCES

1. Sack, T., Gibson, J. G., & Buckley, E. S. The preservation of whole ACD blood Collected, Stored and Transfused in Plastic Equipment. Surg., Gynec., & Obst. *95*: 113 (1952).
2. Muether, R. O., Sommer, A. J., & Koster, B. Studies on Blood Stored in Plastic Bags. Am. J. Clin. Path. *23*: 818 (1953).
3. Knutson, K. P. Plastic Blood Containers. Am. J. Clin. Path. *23*: 813 (1953).
4. Wall, R. L.; Shinowara, G.; Bouroncle, B.; DeLeeuw, N. K. M., & Doan, C. A. An Evaluation of the Preservation of Human Blood Stored in Experimental Plastic Containers: *In vitro* Studies. J. Lab. & Clin. Med. *42*: 665 (1953).
5. Wall, R. L., Buckley, N. M., & Doan, C. A. An Evaluation of the Preservation of Human Blood Stored in Experimental Plastic Containers: *In vitro* Studies. J. Lab. & Clin. Med. *42*: 674 (1953).
6. Strumia, M. M. The Preservation of Blood for Transfusion. Blood *9*: 1105 (1954).
7. Strumia, M. M., Colwell, L. S., & Ellenberger, K. The Preservation of Blood for Transfusion: I; The Effect of Plastic Containers on Red Cells. J. Lab. & Clin. Med. *46*: 225 (1955).
8. Storck, J., & Ardry, R. Colorimetric Micro Determination of Hemoglobin in Biological Media. Ann. Biol. Clin. *9*: 197 (1951).
9. Strumia, M. M., Colwell, L. S., & Dugan, Ann. The Preservation of Blood for Transfusion: III, Mechanism of Action of Containers on Red Blood Cells. J. Lab. & Clin. Med. *53*: 106 (1959).

CONTRÔLE DE LA PURETÉ DES SUBSTANCES ANESTHÉSIQUES[1]

J. G. PATOINE, M.D., et J. C. POULIOT, M.D.[2]

LA NÉCESSITÉ d'administrer les médicaments les plus purs possible est evidente. A plus forte raison, les substances anesthésiques doivent-elles être exemptes d'impuretés puisqu'elles s'adressent à des individus dont la résistance est plus ou moins ébranlée soit par la maladie en cause, soit par le choc, soit par l'agression chirurgicale. Les impuretés des substances anesthésiques, si elles ne compromettent pas toujours directement la vie du patient, réduisent pour le moins la valeur de l'anesthésie.

La plupart des anesthésistes ne disposent ni des moyens techniques ni de la formation requise pour contrôler personnellement la pureté des substances anesthésiques. Après avoir exposé notre opinion sur ce sujet, nous voulons rappeler à votre mémoire quelques tests de pureté facilement réalisables et surtout, signaler afin de les empêcher, les processus de contamination des substances anesthésiques.

Les impuretés peuvent y apparaître en deux occasions: soit pendant la fabrication, soit pendant la conservation ou l'usage, par dégradation intrinsèque du produit ou par apport extérieur (1).

Il est assez rare que les substances anesthésiques en général soient le résultat de réactions chimiques simples. Leur fabrication nécessite souvent la présence de catalyseurs et le résultats en est fréquemment non pas un seul mais plusieurs produits qu'il faut séparer les uns des autres. D'autre part, il est pratiquement impossible pour les anesthésistes et les hôpitaux d'effectuer la vérification à ce stage. Il semble alors suffisamment prudent de se fier à la garantie d'un fabricant de bonne réputation, reconnu pour employer les meilleurs produits bruts; il entretient son propre laboratoire de vérification et observe les standards gouvernementaux requis.

La détérioration des substances anesthésiques pendant la conservation et pendant l'usage nous intéresse davantage parce que nous sommes souvent à même de la retarder sinon de l'empêcher. Elle survient par suite de l'action des agents physiques: air, lumière, température, humidité; ou par transformation chimique tel que oxydation, réduction hydrolyse, polymérisation, tautomérisation ou par combinaison d'un phénomène chimique et d'une activité physique (2).

Lors de son oxydation, un corps s'ajoute de l'oxygène ou perd de l'hydrogène; dans un cas comme dans l'autre, il y a perte d'électrons. La réduction est le phénomène inverse et la substance concernée s'enrichit d'électrons; ces deux phénomènes sont habituellement couplés. L'hydrolyse, c'est la décomposition d'une substance par ou en présence d'eau avec formation habituelle d'un acide et d'un alcool (3).

[1]Travail présenté à la réunion annuelle de la Société Canadienne d'Anesthésie le 5 mai, 1959.
[2]Du service d'anesthésie de l'Hôpital de l'Enfant-Jésus, Québec, P.Q.

La combinaison entre elles de deux molécules ou plus d'un même corps chimique pour former une molécule plus lourde s'appelle polymérisation. Les aldéhydes possèdent cette tendance. Les cétones possèdent une autre propriété: la tautomérisation: c'est le transfert d'un atome d'une position à une autre à l'intérieur d'une même molécule pour former un isomère (4).

Signalons aussi qu'un solide a moins tendance à se modifier qu'un liquide et ce dernier moins qu'un gaz (6).

Comme nous l'avons déjà dit, les normes de pureté des substances anesthésiques sont déterminées par des publications officielles et elles peuvent être recherchées par des moyens physiques ou chimiques (1). Cependant, il faut admettre que les tests simples d'identification sont à peu près inexistants. Il faut presque toujours combiner plusieurs tests d'élimination, ce qui permet d'identifier les substances par déduction.

Les principales propriétés physiques qui peuvent servir à établir le dégré de pureté sont: les points d'ébullition, de liquéfaction, de solidification, la recherche des résidus solides, la turbidité, la densité, l'indice de réfraction, la polarisation, la viscosité. Les moyens chimiques sont les pouvoirs de solubilité, de réduction, d'oxygénation, d'hydrolyse, d'halogénation et le pH. Enfin, des tests biologiques pourraient être utilisés pour compléter l'information.

Certaines de ces propriétés chimiques se manifestent par la formation d'un précipité ou par le changement de couleur d'un réactif. Ces phénomènes sont à la base des tests de puretés auxquels nous faisions allusion.

Oxygène

La pureté de l'oxygène employée en anesthésie doit être de 99 pour cent en volume d'oxygène. De plus, les ballons ne doivent contenir ni CO, ni CO_2, ni halogènes. Etant donné que les normes de pureté exigées pour l'oxygène industriel sont supérieures à celles de l'oxygène médical, il n'y a pas de risque de confusion. Enfin, l'oxygène étant un gaz simple, il ne se décompose pas dans les conditions ordinaires de conservation.

Protoxyde d'azote

Préparation

Le protoxyde d'axote se prépare habituellement en chauffant du nitrate d'ammonium à 240° C. (5). C'est le premier d'une séries d'oxydes d'azote où cet élément a une valence de plus en plus élevée. La toxicité augmente proportionnellement à la valence de l'azote en combinaison avec l'oxygène. Or, ces oxydes peuvent se former en cours de fabrication. Lorsqu'ils sont inhalés, ils se combinent avec l'eau tissulaire pour former des acides toxiques pour l'épithélium alvéolaire. La manifestation clinique en est un œdème pulmonaire.

D'autre part, l'oxyde nitrique (NO) peut se combiner avec l'hémoglobine d'une manière préférentielle, à la façon du monoxyde de carbone (CO) pour ainsi empêcher le transport de l'oxygène.

Si le protoxyde d'azote contient de l'eau, il en résulte deux inconvénients: (i) l'eau se condense sur les valves des appareils lors de la détente, et modifie le débit gazeux (7); (ii) l'eau contribue à la formation d'acides en présence de peroxydes.

Dépistage

Nous disposons de deux tests:

Test au sulfate ferreux (*Fe* SO_4). Il s'agit de faire passer un échantillon du gaz dans une solution de sulfate ferreux. Le présence d'oxyde nitrique se manifeste par une coloration noire due à la formation de nitrosulfate de fer. Fe SO_4 NO). Cependant, cette réaction est réversible et la coloration peut disparaître après un certain temps (Transparent 3-4).

Test à l'iodure de potassium. On peut déceler les oxydes supérieurs au protoxyde d'azote en faisant barbotter un specimen dans une solution d'amidon et d'iodure de potassium 15 M contenant une goutte d'acide acétique glacial. Les peroxydes oxydent l'iodure de potassium et l'iode qui s'en libère produit une coloration bleue en présence de la solution d'empois d'amidon. Ce test présente l'avantage de déceler tous les peroxydes, tandis que le précédent se limite à l'oxyde nitrique.

Retenons enfin, que le protoxyde d'azote ne se polymérise pas et ne se transforme pas spontanément en d'autres produits. La pharmacopée américaine exige qu'il soit pur à 95 pour cent.

Cyclopropane (triméthylène)

Préparation

La préparation du cyclopropane exige plusieurs étapes. Disons simplement que la dernière de ces étapes consiste à faire chauffer du 1 chloro-3 bromo propane en présence de zinc. D'après les exigences de la pharmacopée américaine, sa pureté doit être de 99 pour cent, en volume.

Impuretés

Le cyclopropane se comporte à la fois comme un hydrocarbone saturé et comme un non saturé. Il résiste bien à l'oxydation et les alcalins ont peu d'effet sur lui. Cependant, il se transforme assez facilement en son isomère, le propylène, surtout en présence d'un catalyseur comme la limaille de fer et à température élevée. On peut aussi trouver dans le cyclopropane, du cyclohexane, de l'azote, du CO du CO_2 et des halogènes complexes: v.g., chlorpropane. Mais le propylène demeure l'impureté la plus susceptible d'être rencontrée car il peut se former aussi bien pendant la fabrication que pendant la conservation.

Dépistage

Propylène. On recherche le propylène en faisant barbotter un échantillon du gaz dans une solution alcaline de permanganate de potassium. L'oxydation du propylène aux dèpens du permanganate de potassium fait passer ce dernier de pourpre qu'il était au brun (8). Cette réaction est aussi indicatrice d'autres oléfines.

Bioxyde de carbone (*Transparent no 2*). Au lieu de rechercher le CO_2 par la méthode classique l'hydroxyde de baryum, nous avons utilisé une solution aqueuse de chaux sodée avec indicateur. Nous y avons fait barbotter le cyclopropane suspect. Comme aucune coloration n'est apparue, nous avons conclus qu'il était exempt de CO_2. En contre preuve, on a fait expirer dans une solution identique des patients anesthésiés au cyclopropane. L'apparition d'une coloration bleutée a indiqué la présence de CO_2 dans les gaz d'expiration.

Monoxyde de carbone. Il faut d'abord préparer une solution d'hémoglobine en ajoutant 2.5 cc. de sang à 50 cc. d'une solution contenant 25 mg. d'ac. tannique et 25 mg. d'ac. pyrogallique. On y expose ensuite 250 cc. du gaz à analyser. L'oxyhémoglobine et l'hémoglobine réduite vont réagir réciproquement avec l'ac. tannique et l'ac. pyrogallique pour former un précipité brun. Si l'échantillon contient du monoxyde de carbone, la carboxyhémoglobine qui en résulte étant plus stable ne réagit pas avec ces acides, et retient sa coloration rosée.

Ether-éthylique

Fabrication

La façon courante de fabriquer l'éther anesthésique consiste à déshydrater par chauffage l'alcool éthylique en présence d'acide sulfurique concentré.

Impuretés

Nous divisions en deux classes les impuretés que nous pouvons retrouver dans l'éther. D'abord, les impuretés de fabrication, notamment l'acide sulfurique, l'alcool éthylique et leurs dérivés. On comprend en effet que des molécules des ingrédients de base puissent passer dans le produit final. Dans le second groupe, nous reconnaissons les impuretés de conservation. Ce sont les peroxydes d'éther, les aldéhydes et plus spécifiquement l'acétaldéhyde. La formation de ces produits de décomposition sera favorisée par l'air, la lumière et la chaleur, tandis qu'elle sera retardée par les métaux lourds, en particulier le cuivre, et aussi par la diphenylamine et l'hydroquinone (9).

On considère comme satisfaisant un taux d'alcool ne dépassant pas 4 pour cent. D'ailleurs, l'alcool présente l'avantage d'élever le point d'ébullition de l'éther, avantage dont on tire profit dans les climats chauds et les hautes altitudes.

L'acide sulfurique dans l'éther déclenche, ou tout au moins favorise en présence d'alcool, la formation de peroxydes qui par la suite cèdent leur place aux aldéhydes. Certains auteurs prétendent que l'éther contenant des ces impuretés est toxique pour les cellules respiratoires ciliées. De toute façon, il est certain que sa puissance anesthésique est diminuée par leur présence. Par contre, d'autres auteurs ont trouvé que 0.5 pour cent d'aldéhyde est sans effet chez les animaux d'expérience et qu'une concentration égale de peroxydes produirait tout au plus une légère irritation gastrique chez l'homme.

Dépistage

pH. L'éther doit avoir une réaction neutre. Si on mélange une égale quantité d'eau distillée et d'éther, les deux phases doivent rester neutres. On devra sus-

pecter la présence d'acide sulfurique si le papier tournesol bleu vire au rouge.

Peroxydes (*Transparent no 1*). Le dépistage des peroxydes se fait de la façon suivante: ajouter 1 cc. d'une solution à 10 pour cent d'iodure de potassium à 10 cc. d'éther. Laisser reposer à la noirceur dans un contenant hermétique durant trente minutes. La présence de peroxyde se manifeste par une coloration jaune due à la libération de l'iode élément. Si l'on veut contrôler le résultat de cette réaction on peut y ajouter quelques gouttes d'une solution d'empois d'amidon. Cette solution en présence d'iode libre prendra une coloration bleu foncé (10).

Aldéhydes. Pour la recherche des aldéhydes on met à profit leur capacité de réduire le mercure de la solution de Nessler. Après avoir agité 20 cc. d'éther et 3 cc. du réactif, on laisse former deux couches. Une coloration jaune de la partie aqueuse (i.e., solution de Nessler) indique la présence d'aldéhydes qui, en quantité suffisante, pourront former un précipité jaune.

Il faut se méfier d'une réaction faussement positive. A la longue, l'alcool contenu dans l'éther s'oxyde aux dépens du mercure. Ce dernier étant réduit pourra se colorer ou encore précipiter.

Chloroforme

Préparation

On prépare le chloroforme par réduction du tétrachlorure de carbone en présence de fer comme catalyseur. On y ajoute 1 pour cent d'alcool éthylique pour fins de stabilisation.

Impuretés

Parmi les impuretés que l'on peut retrouver dans le chloroforme, signalons l'acide chlorydrique, certains aldéhydes, de l'acétone, et enfin un dérive cétonique très dangereux, le phosgène.

Dépistage (*Transparent no 5*)

L'eau distillée, après lavage de chloroforme exempt des impuretés ci-haut nommées, restera neutre au papier tournesol. Les corps cétoniques, le phosgène et les aldéhydes produiront une réaction positive en présence de la solution de Nessler.

Si l'on soupçonne la présence d'halogènes, on pourra les faire précipiter par une solution acidifiée de nitrate d'argent.

Trichloréthylène

Les produits de décomposition du trichloréthylène sont les mêmes que ceux du chloroforme, et nous pouvons recourir aux mêmes tests de dépistage.

Discussion

Dans ce court exposé, nous n'avons considéré que les anesthésiques les plus usuels. Depuis plusieurs mois, nous avons pratiqué à maintes reprises les tests

ci-haut décrits. Nous n'avons pas réussi à trouver d'impuretés dans les différents échantillons.

C'est dans le but d'illustrer les différents tests que nous avons contaminé volontairement certains de nos échantillons. Même si à date nous n'avons pas décelé d'impuretés, nous nous proposons de continuer à pratiquer ces tests à l'occasion, ne serait-ce que pour garder en mémoire les principes suivants:

TABLEAU I

LES IMPURETÉS QUE L'ON PEUT RENCONTRER DANS LES PRODUITS ANESTHESIQUES LES PLUS EMPLOYÉS

	Impuretés									
Agents	Peroxydes	Aldéhydes	Acétylène	Hydro-carbures halogénés	CO_2	CO	Oléfines (propy-léne)	Phosgène	HCl	NO (peroxydes d'azote)
Diethyléther $(C_2H_5)O(C_2H_5)$	X	X								
Divinyléther $(C_2H_3)O(C_2H_3)$	X	X	X							
Protoxyde d'azote N_2O										X
Triléne C_2HCl_3		X						X	X	
Chloroforme $CHCl_3$		X		X				X		
Cyclopropane C_3H_6				X	X	X	X			

1. L'éther, le chloroforme et le trilène sont les substances dont la conservation exige le plus d'attention car leur mode d'administration nécessite l'exposition aux agents physiques. C'est ainsi que l'on doit considérer comme suspect l'éther qui a séjourné dans une boite ouverte depuis une semaine ou dans un vaporisateur depuis deux jours.
2. Le cyclopropane et le protoxyde d'azote sont relativement stables pendant leur conservation à condition d'avoir été livrés purs.
3. Les produits de décomposition les plus dangereux sont: le phosgène, l'oxyde nitrique (et peroxydes d'azote), les aldéhydes.
4. En plus de leur toxicité, les peroxydes d'éther comportent certains dangers d'explosion: les vaporisateurs devraient être nettoyés fréquemment car des peroxydes peuvent y demeurer après évaporation de l'anesthésique.
5. L'odeur inusitée d'une substance doit faire craindre la présence d'impuretés.
6. Il ne faut pas hésiter à rejeter une substance douteuse.

CONCLUSION

Nous avons voulu faire une brève revue des principales impuretés qu'on peut rencontrer dans les substances anesthésiques les plus employées.

Il est étonnant de constater qu'avec six ou sept réactions, on peut déceler la majorité des impuretés susceptibles de se former. S'ils ne sont pas hautement spécifiques, ces tests ont l'avantage d'être fort simples tout en ayant une valeur suffisante pour commander l'abandon d'une substance à réaction positive. Ils permettent ainsi d'augmenter la marge de sécurité de l'anesthésie moderne.

TABLEAU II

Impuretés	Réactifs	Résultats positifs
Acidité	Papier litmus bleu	Rose
Peroxydes	KI+amidon	Bleu
CO_2	Sol. de chaux sodée	Bleu
NO	KI (15M)	Bleu
R—CHO	Solution de Nessler	Vert
Oléfines	$KMNO_4$	Brun
CO	Hémoglobine+ ac. tannique / ac. pyrogal.	Rose

Summary

During the last few months we have had the opportunity to check the purity of the following anaesthetics: nitrous oxide, cyclopropane, ether, chloroform, trichloroethylene. We have taken advantage of this to review the principal tests used in this regard. Up to now, we have not found any impurities in the samples examined. However, we have on purpose contaminated some of these in order to prove without doubt the results of the principal reactions.

Depending on the individual anaesthetic, peroxides, propylene, carbon monoxyde, carbon dioxide, aldehydes, and phosgene are the impurities most likely to be found. It is possible with a few simple tests to detect most impurities. We believe that the frequent practice of these tests would widen the security margin of modern anaesthesiology.

BIBLIOGRAPHIE

1. Adrian, J. The Chemistry of Anesthesia. Springfield, Ill.: Charles C. Thomas (1954).
2. Adriani, J. The Pharmacology of Anesthetic Drugs. Springfield, Ill.: Charles C. Thomas (1956).
3. Wertheim, E. Introductory Organic Chemistry. Philadelphia: Blakiston (1946).
4. Gagné. Traité élémentaire de de chimie. (1955).
5. Lee, Alfred. Synopsis of Anaesthesia. Wright (1957).
6. Beckman. Drugs: Their Nature, Action and Use. Philadelphia: W. B. Saunders (1958).
7. Macintosh & Mushin. Physics for the Anesthetist. Springfield, Ill.: Charles C. Thomas (1958).
8. Burger, O. K. Tests for Impurities in Cyclopropane. Anesth. & Analg. *4*: 207 (July–Aug. 1937).
9. Gold, H., & Gold, D. The Stability of U.S.P. Ether after the Metal Container has been Opened: With Preliminaryy Results of a Clinical Comparison of U.S.P. Ether in Large Drums with Ether in Small Cans Labeled "For Anesthesia." Anesth. & Analg. *2*: 92 (March–April 1935).
10. Schukys, J. G., & Neeley, A. H. Studies on the Formation and Decomposition of Ether Peroxides. Anesthesiology *19* (5): (Sept.–Oct. 1958).

MEPERIDINE IN ROUTINE PAEDIATRIC ANAESTHESIA[1]

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MEPERIDINE SUPPLEMENTATION of thiopental, nitrous oxide anaesthesia in adults is widely used today and has been practically routine in our hospital for several years (1). The purpose of the present paper is to report on its use in children and to show that they need much larger dosage of meperidine than might be expected.

Some of the advantages gained from meperidine are: a non-explosive anaesthetic, less tendency to bronchospasm, fewer cardiac irregularities and, particularly, a quiet and early awakening. An analysis of 100 post-anaesthetic recovery room records in recent consecutive unselected adult cases (Table I), which included

TABLE I

POST-ANAESTHETIC RECOVERY ROOM COURSE WITH AND WITHOUT MEPERIDINE

Cases	Anaesthetic	Awake immediately (%)	Average awakening time of remainder (mins.)	Restless (%)	Cried (%)	Restless and cried (%)	Total restless and/or cried (%)	Vomit (%)	Croupy (%)
100 adults	Thiopental, meperidine, N_2O/O_2, Mecostrin, or Flaxedil	71	23	3 (mild)	0	0	0	9	0
200 children (ages 3–13 inclusive)	Thiopental, meperidine, N_2O/O_2, Mecostrin, or Flaxedil	42	20		4	1	8	4.5	0
100 children (ages 3–13 inclusive)	Thiopental, N_2O/O_2, relaxant	29	24	22	7	17	46	18	4
100 children	Vinethene or Vinamar, di-ethylether, N_2O/O_2	91	22	10	19	10	39	20	5

NOTE: Practically all patients were intubated. "Awakening time" is when the patient makes a coherent response to the spoken word.

thirty-six laparotomies and ten vaginal operations, revealed that there were no patients with a systolic blood pressure below 100 mm. Hg during surgery and only two who had a systolic pressure under 90 at any time in the recovery room. No vasopressors were employed at any time and blood was used conservatively. Five patients were given blood in the recovery room. Nine patients vomited a total of fourteen times and three were noted to have been "restless," but required no treatment.

It seems logical to apply this technique to children as their tolerance of hypnosis and pain is less than that of adults. The restless, crying, or screaming children not infrequently seen in the immediate postoperative period are not only hurting themselves but are also keeping the nursing staff busy and distracted, and dis-

[1]Presented to the Western Divisions, Canadian Anaesthetists' Society, Saskatoon, March, 1959.
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turbing other patients. The incidence of what sometimes amounts to "emergence delirium" is greater now that more and more children receive an intravenous anaesthetic which often does not include an analgesic (Table I). The old-fashioned ether anaesthetic provided some postoperative analgesia. This restlessness, nearly always due to pain or residual curarization, is aggravated in many instances by the hypnotic action of the barbiturate which reduces what control the child may have developed. Residual curarization is easily recognized by the short jerky movements of the extremities, due to relatively ineffectual attempts to move, along with a slack jaw and drooping eyelids. Adult patients who have experienced this feeling of weakness or inability to move state that it is very unpleasant.

The administration of adequate doses of meperidine with the anaesthetic, as well as providing analgesia, allows a considerable reduction in the thiopental dosage as well as a reduction in the dose of muscle relaxant, so that the three factors mentioned, pain, hypnosis, curarization, which lead to crying and restlessness are, at least partially, overcome. Since 1952, all my major intravenous anaesthetics have included intravenous meperidine as a preliminary.

Generally speaking, children under nine have received sodium seconal gr. ss to gr. i ninety minutes preoperatively and atropine gr. 1/150 on the stretcher "on call." Those who would not take the seconal orally were given it per rectum after perforating the capsule. Rectal seconal capsules are quite ineffective and the route used was often unknown except by the sedated or unsedated state of the patient. Those who received it orally would need less thiopental. Children aged nine to thirteen usually received meperidine 20–50 mg. with atropine 1/150 gr. one hour preoperatively. Those who received meperidine, but no barbiturate, would need somewhat more thiopental and possibly a little less meperidine with their anaesthetic.

The only contraindications to an intravenous anaesthetic were asthma, inaccessible veins, or a marked lack of patient co-operation. Asthmatics were always given ethyl vinyl ether or vinyl ether/di-ethyl ether/nitrous oxide anaesthetic (2). In practice almost all children six years of age or over received an intravenous anaesthetic. About 50 per cent of the five-year-olds were anaesthetized in this manner, but only a few under five.

Technique

The head of the operating table must be slightly lowered to prevent hypotension. One nurse holds the child's arm while another holds his head to the opposite side and distracts his attention until the venupuncture is accomplished and the patient asleep. The puncture is made with a #22 or #20 needle attached to a 2 cc. syringe containing the meperidine. At least two minutes is taken to administer the meperidine followed by the relaxant (Mecostrin[3] or Flaxedil[4]) which is given in a few seconds. The thiopental syringe is now attached and an estimated dose given (3–5 cc. of 2 per cent). The patient's lungs are inflated

[3]Tubocurarine dimethyl ether chloride.
[4]Gallamine triethodide.

with oxygen for about half a minute and then a little more thiopental is usually given (2–3 cc. of 2 per cent) and the intubation is performed. The anaesthetic is then continued with nitrous oxide–oxygen 6/2 L. per minute with either a circle system or a modified Ayres T-tube technique. Respirations are controlled or assisted as necessary. This is usually all that is required for short operations, for example, tonsillectomies. If the procedure lasts longer than thirty-five minutes, increasing the nitrous oxide–oxygen percentage to 8/2 L. per minute will usually give about ten minutes more of adequate anaesthesia. In longer procedures, 12–25 mg. of meperidine may be given intravenously as needed. If an intravenous is not running, then a little cyclopropane or ether is used. If the procedure is finished in under twenty-five minutes and/or the jaw or abdominal muscles are slack, 0.5 mg. of Prostigmine is given slowly intravenously or intramuscularly while the patient breathes 100 per cent oxygen. Opiate antagonists have not been found necessary but could be used, for example, 1 mg. Lorfan®[5] if the respiratory rate is unduly low at the end of operation.

Meperidine is not usually used if the procedure is expected to take less than twenty-five minutes.

Dosage

Table II gives the average of the doses actually used in 332 consecutive patients. The apparent discrepancy in the case of the four three-year-olds is because these were long operations and a little heavier dosage during induction would not leave a residual effect at the end. Early in the series the dosage of

TABLE II

Average Doses of Drugs Used Intravenously for Intubation for Various Procedures in 332 Consecutive Cases

Age	Cases	Meperidine (mg.)	Thiopental (2 per cent) (20 mg./cc.)	Mecostrin (1 mg./cc.)	Flaxedil (20 mg./cc.)
3	4	32	6	—	1.5
4	14	40	5	2	1.3
5	29	47	5.7	2.3	1.7
6	64	54	5.8	2.7	2
7	52	58	6.1	3	2
8	52	60	7	3.7	2.4
9	35	61	7	4	3
10	27	62	7.6	4.4	3
11	19	68	7.7	4.6	4
12	19	63	11	4.9	4
13	17	66	11	5.6	4

meperidine was too low for the desired effect (25–50 mg.) and later it was too high in some cases (75–100 mg.). For the last three years it has been around 40–70 mg. which has proven very satisfactory in the great proportion of cases. Individual variations in reaction to drugs exist of course, but one can judge the dose of meperidine to a great extent by the age, sex, size, and vigour of the patient, as well as by the effect of the slowly administered drug. The relaxant dosage is necessarily a pure estimate based on the above factors and the dose

[5]Levallorphan tartrate.

of thiopental is just a little more than a sleeping dose, which is usually added to immediately before intubation or, occasionally, immediately afterwards.

Complications

With the dosages of meperidine indicated in the table and given slowly, the child becomes a little sleepy, the pupils contract, and many complain of an itchy nose. Breathing remains adequate and the colour is unchanged. With too large a dose the child may suddenly feel that he is falling and tend to panic, throwing the free arm up and grasping. If this happens, the relaxant and thiopental are given quickly and the patient intubated after oxygenation. There have been no ill effects from this. A little less than 1 per cent of patients complained of nausea near the end of the meperidine injection and another 1 per cent vomited; 0.5 per cent complained mildly of being dizzy; 1 per cent complained of some epigastric pain. Some of these patients had received too much meperidine too fast. The blood pressure was usually not taken but in one case where hypotension occurred it was soon corrected by lowering the head of the table which had inadvertently been left a little elevated. When any of the undesirable features noted above occur, the meperidine injection is terminated and the induction completed.

On two occasions the needle came out of the vein after the relaxant had been given. One case was given cyclopropane and intubated and the other nitrous oxide oxygen while another vein was found. Both children were curarized and unhappy, but neither one remembered the incident, the meperidine having produced amnesia.

Postoperative Course

The following remarks are based on 200 consecutive cases for which recovery room notes are available (Table I). Of these, 84 patients (42 per cent) were awake on arrival in the recovery room and the remaining 116 were "awake" in an average of twenty minutes. Some of these latter were simply dozing and could easily have been aroused earlier. Eight (4 per cent) patients were noted to have cried and six (3 per cent) to be restless at some time. Two were both restless and crying. Nine (4.5 per cent) vomited. The respiratory rate was nearly always in the range 18–24 per minute. Only one patient had a respiratory rate as low as 14 per minute on one occasion. The total of 8 per cent, who were restless and/or crying contrasts with 46 per cent of those who received an intravenous anaesthetic without meperidine and 39 per cent who were given an ether–nitrous oxide anaesthetic.

Restlessness and/or crying was usually due to a residual curarization, too little meperidine or an excess of barbiturate (thiopental and/or seconal). Residual curarization, easily recognized by short sharp jerky movements of the extremities, was corrected by Prostigmine 0.5 mg. intramuscularly or intravenously. An excess of barbiturate, sometimes due to too much seconal preoperatively, would correct itself in time.

Two children (ages 8 and 6), who were bleeding from the tonsillar fossa, were controlled by digital pressure with an adrenalin-impregnated sponge. They co-operated very well and did not appear to be feeling pain. These patients would otherwise have been returned to the operating room for haemostasis under anaesthesia.

There were no cases that gave cause for alarm postoperatively. On the contrary, it was very satisfying to see the child awake, quiet, and co-operative in the great majority of instances. It should be mentioned that in our post-anaesthetic recovery room all children have the foot of their cribs elevated to a 30° angle. The beds are levelled when they regain consciousness.

Discussion

This method was developed in an attempt to have a quiet, safe immediate postoperative course as well as a smooth induction and maintenance. It has been used on all indicated cases for the last six years and will be used until something better comes along.

One advantage is the excellent conditions for intubation and the lack of reaction to or "bucking" on the tube. It is ideal for teaching interns, who are not rushed. The lack of bucking during intubation and the smooth course of the anaesthetic probably protect the vocal cords. There were no laryngeal reactions in these cases.

The apparently large doses of meperidine used may be surprising, but it seems that children tolerate and metabolize it quickly. In fact in the longer procedures, for example, mastoids and laparotomies, it was nearly always necessary to give another dose of meperidine (12–25 mg.), thirty-five to forty minutes after induction. Doses indicated in the table are not used preoperatively but only in the operating room with all facilities present. Also, when meperidine is used after an ether induction, much smaller doses are given (for example, 5–15 mg.) and repeated when necessary.

Another asset of this form of anaesthesia is noted when doing cystoscopies and retrograde pyelograms in children. The respirations are slow, thus facilitating X-ray examinations.

The sequence meperidine–relaxant–thiopental must be adhered to. Intravenous meperidine develops its peak action in about four minutes, the relaxant in one to two minutes and the thiopental in fifteen to twenty seconds, so that all drugs are at practically maximum effect for intubation. Intubation was invariably simple and bucking or straining on the tube absent. The course of the anaesthetic is very smooth also. The successful suturing of a badly lacerated eye which was leaking vitreous was certainly facilitated by this method. In this type of case, where vomiting or straining is a great hazard, promethazine 6–12 mg. should be given prior to the meperidine.

In conclusion, reflex apnoea should be mentioned in relation to this technique. The commonest cause by far of apnoea with this technique, and with some others, is reflex. The presence of the endotracheal tube, the artificial respiration, and the lack of afferent impulses, all combine to produce this effect. Moving the

tube or turning the patient at the end of the operation will always initiate respiration.

Conclusions

The restless and crying children in the modern post-anaesthetic recovery room have been attributed to hypoxia during the anaesthetic and to psychological causes. There is no doubt that in the great majority of cases these little patients are suffering from the pain of acute recent trauma, the hypnotic effect of barbiturates, or residual curarization. The use of meperidine intravenously, as part of the anaesthetic, reduces the incidence of these unpleasant sequelae markedly, as well as providing smooth induction and maintenance and early awakening from anaesthesia.

REFERENCES

1. Walton, F. A. Nitrous Oxide–Demerol–Mecostrin Anaesthesia. Proc. Canad. Anaesth. Soc. 2: 119 (1953).
2. Walton, F. A. Bronchial Asthmas and Anaesthesia. Canad. M. A. J. 74: 624 (1956).

RESUSCITATING DEVICES AVAILABLE TO THE LAYMAN[1]

D. R. Collins, M.D., and M. H. Schultz, M.D.[2]

We have found it difficult to reduce resuscitation to plain terms beyond dispute. As anaesthesiologists, we may possibly think we have the last word on this subject, but take away our equipment and consternation might be the kindest word that could be applied to our state of mind. And, if the last word on this subject goes to us, then certainly the first word must go to first-aid personnel. However, their position is, perhaps, even more difficult than ours, for they are presented with the controversy of manual *versus* mechanical means, further confounded by the question of which manual or which mechanical means.

The Oxford Dictionary at least is straightforward. It says that resuscitation is the act of restoring to life. The first-aid manuals qualify this definition by stating that it is the prevention of death by artificial or mechanical respiration. They qualify still further: for successful resuscitation there must be an adequate supply of oxygen for the vital centres, the respiratory centres in the brain and the heart. Finally, two authorities on this subject, Ivy and Schwerma (1) of the Illinois School of Medicine, have laid down these absolute qualifications:

There must be:

(1) An unobstructed airway.

(2) An adequate functioning alveolar surface for the absorption of oxygen and the elimination of Carbon Dioxide.

(3) Circulating blood to transport the absorbed oxygen to the heart and central nervous system.

(4) A myocardium and central nervous system still capable of being resuscitated.

Once the airway is cleared, artificial respiration should be commenced at once with the most efficient method available. If no apparatus is immediately available, the manual method must be used. When a resuscitator arrives, it can always be substituted.

And so with this advice they leave us with vague notions of push and pull machines that seem efficiency itself, but about which we have our doubts.

Manual Methods

Comparing the tidal air exchange of the various manual methods of resuscitation we find: Schafer, 340 cc.; Sylvester, 400 cc.; Eve, 560 cc.; Mouth-to-Mouth 970 cc. The Schafer method involves alternating push and release on the lower thoracic cage in the prone position to achieve tidal air exchange. The Sylvester method involves pull on the arms and release in the supine position. Both, we believe, rely too much on the elastic recoil of the chest wall which may not be adequate, and we cannot be absolutely certain of that all-important unobstructed

[1]Presented at the Western Divisions' Meeting, Canadian Anaesthetists' Society, Saskatoon, Sask., March 20, 1959.

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airway. The Holger Neilsen method is a combination of these two and is called the push and pull method.

This subject of resuscitation is as old as time itself. In the Bible, in Kings I and II, it is revealed that Elisha revived the Shunamite child by mouth-to-mouth breathing; Hippocrates is said to have suggested that respiration might be restored by inserting a tube into the trachea and forcing air down it; in the Middle Ages, Paracelsus and Vesalius used the fireside bellows to inflate the lungs. The modern history of artificial respiration began about 1775 when John Hunter invented a double action non-rebreathing bellows which was adopted by the Royal Humane Society of London, while the Dutch Humane Society advocated mouth-to-mouth insufflation for victims of drowning. The Navy rolled them over a barrel. The nineteenth and early twentieth centuries produced many methods of manual respiration of which the best known are the Sylvester, the Schafer, and the Holger Nielsen.

This last method is now officially recommended by all life-saving organizations. The advantages are that the prone position of the patient with the head to one side should ensure a good airway. It is a push-pull method and not only is pressure applied to the upper chest to induce expiration, but the arms are lifted to induce inspiration. Therefore, there is increased efficiency of pulmonary ventilation and the lack of pressure on the abdomen reduces the risk of inhaling regurgitated water.

For the Eve Method, a rocking stretcher is necessary. The respiratory exchange is effected by changes in the position of the diaphragm as the patient is placed alternately in a head-downward and a head-upward position. The advantage of this method is that it affords a large respiratory exchange and increases cardiac output. It also promotes drainage of fluid from the chest. The ever water-conscious Navy changed from the roll over the barrel method to the rock of the Eve stretcher, firm in the belief that they were getting "the water out of the lungs," as well as inducing respiration. Since drownings are the most common accidents we are faced with, let us consider this "water in the lungs."

Dr. H. G. Swann reported on fresh water drowning in 1949 (2) that when an animal is totally immersed there is an initial period of struggling and apnoea. Within two minutes, an involuntary inspiration occurs and water is drawn into the lungs, usually in large quantities. There is an immediate, enormous absorption of this fluid into the circulation across the alveolar capillary membrane. An amount of water equal to 60 to 150 per cent of the blood volume can enter the circulation in a few minutes. The result is twofold.

(i) Haemolysis with potassium release and great increase in the K/Na ratio occurs. The coronary circulation receives this highly abnormal blood with serious biochemical insult to the myocardium, as well as anoxia.

(ii) There is great overloading of the circulation. The result is ventricular fibrillation in from three to seven minutes. The onset occurs almost simultaneously with cessation of effective respiratory excursions.

In 1951, data revealed that nineteen out of twenty victims of drowning have water in the lungs and haemodilution. Electrolyte studies showed changes capable of precipitating ventricular fibrillation in at least half.

In salt water drowning there is no change of the K/Na since there is no haemolysis. There is, however, haemoconcentration with movement of water from the blood into the lungs. The heart failure is due to anoxia and not to fibrillation. Water in the lungs can kill not only by causing anoxia and biotoxicity at the time of the accident, but also by inducing severe pulmonary oedema in both types later, the cause of death being ascribed to pneumonia or exposure. This pulmonary oedema is probably the reason why in many cases of successful resuscitation the victim succumbs hours, or even days later.

Time and tide are, therefore, decisive. Respiratory failure occurs some time before irreversible circulatory failure in one-third of cases, in one-third during failure, and in one-third after failure of circulation (on experimental evidence); so we must get busy immediately—and the Navy rule is right—we must try to "get the water out." Manual methods employing the prone position would, therefore be best: Schafer and Holger Nielsen, when other equipment is lacking. Nevertheless, the skill of the rescuer is so very important in maintaining a rate of at least fifteen per minute and tidal exchange that we must look to simpler and better methods. The American Red Cross and the Council of Physical Therapy state that mouth-to-mouth resuscitation offers the greatest scope for general use and success (4). This method employs the basic physiological principle of using expired air as the ventilating gas. With normal respiration, expired air contains approximately 16 per cent oxygen. In resuscitating a drowning victim, the first 150 cc. of the rescuer's expired air which is dead space air and not involved in alveolar exchange is the first exhaled into the victim's lungs. So that the dead space air may have a nearly atmospheric composition, the rescuer should always take an inspiration just before each insufflation. The chief advantages of this method are: airway obstruction can be noted and dealt with quickly; less energy is expended; the procedure can be continued for hours; no equipment is needed. However, the effectiveness and facility of this method can be improved by the use of an ordinary anaesthesia face mask, airway, or endotracheal tube. The modification of two airways affixed end to end (Safar airway) is a useful adjunct. An infant and an adult size airway should be kept on hand.

The nose of the victim should be blocked when this technique is used on an adult and his head maintained in a well-extended position with the jaw pulled forward. In infants, one may insufflate through the nose and mouth after following the carefully prescribed steps to clear the airway. In tiny babies, Dr. Apgar recommends insufflation with small puffs of air from just the mouth and cheeks. The rate should be at least twenty per minute, whereas in adults twelve to fifteen per minute is adequate and, if correctly done, tidal volumes even over 1,500 cc. can be attained.

Mechanical Methods

So far, we have been on safe ground since we all know that to do something intelligent during those first moments in an emergency is beyond reproach. However, when it comes to carrying on with mechanical devices, the thought searching begins. Even though a good mechanical resuscitator was brought out by Erickson

and Johnstone in 1927, it was not until 1950 that the Council on Physical Medicine and Rehabilitation of the American Medical Association published its tentative minimal requirements for inhalators and resuscitators. However, it had approved of the E & J in 1939 and the Emerson in 1942 (5) in principle. Let us consider these two types.

Several models of each are available, but all have the same type of head which allows them to be used as a resuscitator or inhalator or aspirator, according to the position of the control lever. The E & J inhalator is used when extra O_2 is needed by the breathing patient. It delivers 7 L. per minute with this advantage over the Emerson that the air/O_2 ratio can be adjusted by the central control valve (7). The resuscitator has a positive phase of 13 mm. Hg and a negative of 9 mm. Hg regulated by the toggle diaphragm in the head. During the inspiratory phase, the flow of O_2 is directed into the lungs until a pressure of 13 mm. Hg is built up. At this time, the toggle diaphragm is tripped and the gas flow is re-directed by the Venturi chamber so that the natural expiratory flow is assisted by a suction effect which pulls the gases out of the lungs until a negative pressure of 9 mm. Hg is reached. The toggle now releases and returns to its original position and the cycle starts again.

Why the negative pressure phase? In 1953, Maloney, Elam, *et al.*, showed that in normal patients there may be a 12 per cent drop in cardiac output with intermittent positive pressure alone, and up to 42 per cent in those with respiratory paralysis or an inadequate circulation. Positive-negative pressure did not impair circulation in either type.

The positive pressures necessary to inflate the newborn baby's lungs is the same as for an adult. The only difference is that it takes a little longer in an adult. The difference in time, therefore, determines the respiratory rate and the volume of oxygen delivered. These pressures are considerably less than a premature infant can develop in its own lungs, and the control valve can be set at slow for very small infants or if there is partial obstruction of the child's airway.

It has never been known to cause lung damage. Though there is a danger of forcing regurgitated fluid into the lungs, we are trying to save a life and there is an aspirator attachment to deal with fluid in the airway.

Other resuscitators worthy of mention are: the McKesson, which operates on a volume adjustment which I believe adds slightly to its complexity; The G.L.B.–Goddard Bennet Lovelace (8) infant hand resuscitator consisting of hand bulb, positive pressure gauge with pressure control, use of which is restricted to medical personnel; last, and, I believe, foremost the A.M.B.U., or Rubens type, which adds to mechanical resuscitators what mouth-to-mouth adds to manual methods, offering a rare combination of simplicity and efficiency (9). The resuscitator is the equivalent of our bag and mask. It has a Ruben's non-rebreathing valve and the bag has a foam rubber lining to make it self-inflating. It can deliver a tidal volume of 400 to 1,000 cc. depending on compression and can be used on infants since on compressing the bag with one hand, a pressure of 60 mm. H_2O will not be exceeded. Oxygen can be vented into the bag. There is a suction pump operated by a foot or hand bellows connected to a suction bottle and can create a vacuum of from 190 to 300 mm. Hg.

Conclusion

In our final evaluation, we would reiterate the first-aid rule, "Don't wait—resuscitate," the best way one knows how, and mouth-to-mouth is probably the best. As soon as a mechanical resuscitator comes to hand, use it.

Clear the airway and try to clear the water out of the respiratory system.

Finally, let us hope we see more good hand resuscitators such as the Rubens available in all places where there is the possibility of drowning, or shock accidents, and that anaesthetists and first-aid personnel will be able to work together more closely in this service.

REFERENCES

1. Ivy, A. C. & Schwerma, Henry. Asphyxia and Resuscitation (July 26, 1952).
2. Donald, K. W. Drowning. Brit. Med. J. (July 16, 1955).
3. Rossiter, Frank. Resuscitation. Arch. Phys. Therap. (Oct. 1942).
4. Council on Medical Physics. Mouth-to-Mouth *vs.* Artificial Respiration. Am. J. Med. (May 17, 1958).
5. Council on Physical Therapy, (May 13, 1939) Am. J. Med. *112*, reprint.
6. Reprint on E & J Resuscitator. Burbank, Calif.: E. & J. Mfg. (March 6, 1958).
7. Reprint on Emerson Resuscitator. Cambridge, Mass.: J. H. Emerson Co. (Dec. 17, 1957).
8. Reprint on G.L.B. Unit. Los Angeles: Bennett Respiration Products, Inc. (1958).
9. Report on A.M.B.U. Unit. Toronto: British Oxygen of Canada Ltd. (Aug. 1958), 2nd ed.

TEN YEARS OF GROUP PRACTICE

ERIC WEBB, M.D., and H. B. GRAVES, M.D.[1]

THE DEVELOPMENTS in Anaesthesiology by Hollywood press agents in the past twenty years would be described as "Colossal, Stupendous, Gigantic." Advances in the basic sciences have been reflected in a greater understanding of anaesthesia and have introduced to our specialty such clinical applications as more adequate premedication, the tranquillizers, the relaxants, controlled respiration, one-lung respiration, replacement therapy, electrolyte control, hypotension, hypothermia, circulatory by-pass with oxygenators, and finally (maybe "finally" is the wrong word) elective cardiac arrest! But, in our opinion, a development just as important as these is the concept that an anaesthetic service to a given community can be rendered more proficiently by anaesthesiologists working as a group than by the same anaesthesiologists working as individuals. The exact terms of reference under which a group operates may differ widely, but, in our opinion, a full partnership offers the greatest advantages with the fewest disadvantages. We wish to discuss our own partnership with a view to elaborating this rather sweeping generalization.

At the Vancouver General Hospital the principle of a closed anaesthetic staff, with patients assigned by the Chief of the Department, was established early in the twenties under Dr. D. D. Freeze's régime. This greatly facilitated organization of the Department in the immediate post-war period. In 1947, under Dr. M. Digby Leigh, the personnel of the Department was added to, the scope and load increased, and the teaching programme was greatly expanded. The hospital charged and collected the fees, and the anaesthetists were on a part-salary, part fee-for-service basis, paid by the Hospital. When the British Columbia Government announced its hospital insurance scheme, to commence in January, 1949, the anaesthesiologists at the Vancouver General Hospital immediately informed the Administrator of the Hospital that as of September 15, 1948, they would bill patients privately, but in all other aspects continue to provide exactly the same service that had been provided previously. The eleven staff members formed the partnership known as "M. Digby Leigh & Associates." When Dr. Leigh left the Group in 1953, the title was changed to "Associated Anaesthetic Services." Of the eleven charter members, four are still with the group, which has grown to include twenty-three members. Early in its history, one member left to help inaugurate a Department of Anaesthesia at Royal Columbian Hospital, New Westminster. Two others left to inaugurate the Anaesthetic Department of the Grace and Burnaby Hospitals. In 1953, Dr. Leigh and Dr. Belton left for the Children's Hospital in Los Angeles. Two others retired. The replacements have been made from various training schools. Fourteen of our partners are graduates

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of our own training programme; two were trained at the University of Toronto; two at McGill; and one received his training in Britain. In the ten years we have been in operation, we have had three men leave before becoming full partners to take up practice somewhere else. At present, the 23,000 cases per year for which we are responsible are looked after by the twenty-three partners of Associated Anaesthetic Services, two part-time salaried employees (one on supervised practice), and a group of ten residents.

The contact of anaesthesiologists with a surgical patient is a four-party situation. In the centre, of course, is the patient, with the surgeon and anaesthetist standing close by. In every instance there is somewhere in the picture the administrator of the hospital. Each of these four stand to benefit greatly from the group practice of anaesthesia.

The Patient

The patient ordinarily has no private anaesthetist whom he wishes to look after him. Under a system which guarantees him the services of a qualified anaesthesiologist, he is safely and comfortably guided through his surgical adventure. If, as occurs occasionally, the patient has a personal preference for a particular member of the group, this is respected and arrangements are made accordingly. Physicians are allowed to request anaesthetists for themselves and their families, but not for their patients. All emergency anaesthesia is performed by a qualified anaesthetist, with or without a resident present. If the patient's condition should warrant two staff anaesthetists, two are assigned.

The Surgeon

All partners are adequately trained in all aspects of anaesthesia and our surgeons are accustomed to having any member of our group in attendance. Some of us are more pleasant to work with than others, but all have the reputation of doing a good, safe, more than adequate anaesthesia. All elective anaesthesia is assigned by the anaesthetist-in-charge. All emergency anaesthesia is booked by the first anaesthetist on call, after consultation with the Operating Room Supervisor. The surgeon has no trouble in "arranging for" an anaesthetist, because when the case is booked, that has been looked after for him. In this day of "possible radicals," exploratory thoracotomies which turn into either marathons or closures, slow surgeons, and particularly in a hospital with as many operating rooms as ours has (eighteen), it is not infrequent for a single operating room to be either hours behind schedule or empty. When a partnership exists, switches in operating room schedules are more easily arranged and no one is upset. Personal income is not involved, and everyone is happy. We try to ensure that any surgeon and anaesthetist who wish to work on a particular problem with regard to patient care may do so. Then the decision reached and the information gained are shared at a staff meeting, and everyone benefits.

For good reasons, which nearly anyone can appreciate, we have created a team which does the open-heart pump-oxygenator cases. These men were subsidized to an extent during the time they were working out their laboratory

efforts, and we feel this has provided better care of these very difficult cases. These cases are, however, the only cases which are "the property," as it were, of any special anaesthetist.

With the patient in the post-anaesthetic recovery room, the surgeon, of course, has his share of the after-care responsibility. But, with so many of us available, the recovery room patients are obviously going to get the early attention to emergencies that make this type of service so valuable. The recovery room nurses are directly responsible to the anaesthetic staff.

Administration

With a group of qualified anaesthesiologists, the administrator of the Hospital can be assured that at all times he has a Department of Anaesthesia of which he can be proud. We are on call for emergency resuscitation needs at any time. Staff anaesthetists are present in the hospital twenty-four hours out of every day, seven days a week, fifty-two weeks a year.

The Anaesthetist

Let us quote verbatim the introduction to our partnership agreement:

Whereas the Parties hereto are agreed in their desire to practice Anaesthesiology in that manner in which is most likely to produce good medicine and good citizenship, and believe with deep sincerity that all practice of medicine should be governed by strict adherence to a code of ethics such as that expressed by the College of Physicians and Surgeons of British Columbia. Now this agreement witnesseth that the Parties hereto have agreed to enter into Partnership for the practice of their profession, specializing as aforesaid, upon the terms and conditions and under and subject to the provisions hereinafter contained.

Further the Parties hereto are agreed that it will be the constant aim of this Partnership to promote and raise the standards of Anaesthesia by diligent care of patients, by attaining knowledge in all ways, and by teaching, both of undergraduate students and other qualified Physicians.

And further, the Parties hereto believe that by joining together in Partnership, all Members can and should share their time in proper proportion between the practice of Anaesthesiology, the study of their profession and specialty, and the care and enjoyment of their families. And also that they can help one another in daily work, ensure freedom from financial worries in time of sickness, and share with each other the costs and efforts of further training, attendance at conventions and meetings and all other things which will bring additional knowledge to the Partnership as a whole. And the Parties hereto believe that all others who, from time to time, may sign this Agreement must be willing to sacrifice aims of self and, in some measure, size of income, to share in and gain from the Association now created.

Working as we do, as a fully interdependant, mutually supporting partnership, we escape many of the galling features of an individual anaesthetic practice. Holidays are arranged well in advance, and do not interfere with our practice. If cases evaporate in the middle of the morning, it is not a catastrophe. Assigned to a morning of staff (charity) tonsils, our income does not suffer, and we are able to spend that morning as enthusiastically as if the cases were all private and remunerative.

When hypothermia started to become popular, we sent, at no personal expense,

two of our group on a tour of several centres which were leading in this field. They returned to our hospital and have shared their experience with the Department. As mentioned previously, several of our men have been semi-subsidized in their efforts to put anaesthesia for open cardiac surgery on a well-founded basis. One of our members is the hospital pump-oxygenator expert, and this, while it has been somewhat costly, has contributed considerably to what we feel is one of the best series of open heart procedures done to date. In all humility, we have built on others' work, but we have also made our own contributions.

We have established a fairly extensive programme for covering our members if sickness overtakes them. During any twelve-month period, three months' sick leave is allowed. For each year with the partnership, a three-week credit is established in the members' sick leave accounts. A partner who has given good, healthy service for fifteen years, is thus allowed forty-five weeks of sick leave if he finally does become ill. During sick leave, all income is continued, except the overtime case allowance, for the first three months, and then a reduced income is available.

We hold a weekly staff meeting as one of our major educational features. At these meetings, theses are read, cases of interest discussed, the newer features of clinical and basic work reported. Journal Club is rotated through the various staff members. This whole approach makes for a continuous postgraduate course for all of us. All in all, for anaesthetists, our partnership has a lot to offer on the positive side.

In order to arrange six weeks' vacation a year for each of twenty-three members, it is necessary to have three men on holidays most of the time. A roster from year to year decides in what order selection of holiday periods is to be made.

We have a large number of evening emergencies, and of course, our usual number of obstetrical deliveries in the night hours. These are covered by the two night anaesthetists, who come on daily at 8:00 P.M. and work until 8:00 A.M. These night anaesthetists have half a week on duty and then forty-eight hours free. They are not on duty in the day time. On call is a third anaesthetist who is assigned a light morning following, or left off the slate if circumstances permit.

A duty roster is maintained, and set up for six to eight weeks in advance. As the afternoon slated cases are finished, the anaesthetists on the lower calls are freed, and the higher call men carry on. The roster is flexible, so that switches in calls necessitated by special circumstances are easily arranged.

Probably the best feature of our operation is the fact that at all times, regardless of financial status, the patient is the direct responsibility of a staff member. We believe this is the proper manner to practise and teach anaesthesia. This does not mean that the residents stand by and watch. It does mean that every patient, however, has a staff anaesthetist personally supervising the administration of his anaesthetic. The arguments may be raised that this makes cowards of our residents; that they do not develop the habit of making their own decisions; that they are not matured. This is not necessarily so, if the supervision is done carefully and if each decision is made on a consultative and educative basis, rather than a "just do as I say" basis.

The financial return of each operation is pooled. Each member of the partnership receives a basic allowance per four-week period, a per hour allowance for work done between 1 P.M. and 8 A.M., and on Sundays and holidays, payable monthly, and the residue is divided as a quarterly dividend. Junior partners purchase their interest by a system of increasing participation in the residual monies. New entrants into the group are on probation for one year. During this time, professional capacity and personal compatability and the acquisition of "group mindedness" are carefully assessed. The professional ability is of no more importance than the compatibility, and might be of even less concern.

Disadvantages

The biggest single disadvantage to our type of practice is that, at all times, we must forget our own selfish ends, and work together as a really unified group. This is difficult for some people, and impossible for others. Self-advertising, selfishness, avoiding responsibility cannot be condoned, and the little things that make actual practice more pleasant, coffee relief, starting an intravenous, lending a helping hand, etc., come to the fore. Each individual has to be co-operative and "group" conscious.

One large area of the anaesthesiologists' field is the provision of service to the obstetrical patient. No one who has ever looked at this problem can come to any other decision than that we as anaesthesiologists, have a great deal to offer, and furthermore, a duty to make our services available in the case room. All is not that simple, however, and it is very easy to appreciate why many obstetrical departments are woefully neglected by their anaesthetists. Tradition, abetted by a lack of education by us of our obstetrical confrères, has kept fees for this work much lower than other anaesthetic fees. The hours are bad; the volume of work is unpredictable; and scheduling of a programme of obstetrical cases is impossible. Daytime cases interfere with surgical work, evening cases interfere with relaxation, and night cases interrupt sleep. Sometimes for hours not a single patient is being delivered, and without warning, three or four mothers are crowning at once! Recognizing our responsibility to the case room, we have come up with an answer to many of these undesirable problems. One staff man with a resident is assigned for a four-week period to the case room to cover the hours from 8 A.M. to 4 P.M. The staff man can initiate the resident to obstetrically oriented anaesthesia, with a certain continuity of approach. Ordinarily, the rest of the group can forget about the case room work for this period of the day, but if work load suddenly gets very heavy, help can be quickly obtained from the general pool of anaesthetists in the other buildings. After 4:00 P.M., the case room calls are covered by the general call group. We have found that while this programme is somewhat expensive to the group, it covers the general obstetrical load with the least bother to us, and gives most effective service to the mother and obstetrician. We have adopted the general philosophy of "anaesthesia for delivery" rather than "prolonged regional anaesthesia for first stage" as our personal and combined attitude. While some may quarrel with this view, we have felt that, economically, prolonged regional anaesthesia presents too many problems, and we prefer to

restrict this type of anaesthesia to cases where it is definitely indicated for medical reasons.

Teaching

Every anaesthetist on this continent, and especially in Canada, can think back to the days when the teaching of our specialty was in the hands of a very small group: Bourne, Griffith, Leigh, Aikenhead, Watts, Stewart, Shields, Robson, Leech, Freez—and no intentional slight is meant if we have omitted your mentor. We have followed in the steps of these men as closely as possible in attempting to spread knowledge of an enthusiasm for modern anaesthesia. Undergraduates spend a week in the Department, usually with one staff man for the whole week, and an attempt is made to show them the broad implications rather than make them proficient in one or two restricted techniques. Interns, on the other hand, are with us for a month, and because some of them are going to be in practices where they must administer some anaesthetics without further training, an attempt is made to give them a high degree of safety and proficiency in several simple and widely applicable techniques, and at the same time provide some idea of the broader reaches of the field.

Many doctors practising in British Columbia are forced by physical factors to give their own anaesthetics, and in many areas the physician saturation is such that anaesthetics are given after scant or no training. We have many applications for short training periods from this group of the profession. We feel that while short training is not as desirable as longer periods of at least a year, there is nevertheless a place for three-month courses for those doctors forced to work in a moderately isolated area.

Since 1947, when the reorganization of the Department took place, we have had sixty-four residents pass through the department: fifty of these have been certified by the Royal College of Physicians and Surgeons, and two have obtained their F.R.C.P.(C). Nine of the fifty certified have qualifications to sit for the F.R.C.P.(C) if they so desire. Twelve were from the United States or withdrew. At present, there are nineteen in the Post-Graduate Course in Vancouver.

Close contact is maintained with the university departments in the basic sciences, and for eight years we have had a fellowship in Basic Science at the University of British Columbia supported by three pharmaceutical firms. The lecture programme for students, interns, and postgraduate courses is divided up among the members of the group so that it does not fall on one or two sets of shoulders, and at the same time, the beneficial effect of preparing a series of lectures is spread around. As pointed out previously, our residents are not used to stock certain departments to relieve the need of staff men, but are in constant contact with one of the staff, who is in charge of the cases that the resident may do.

We may have created an impression that Associated Anaesthetic Services not only "knows all the answers," but in addition has a "corner on the answers." This, we regret to say, is not true. In ten years of existence, we have gone up many blind alleys. We have been lost at times in a maze which, to our surprise, led us

out to a position we had occupied several years previously. Many of our habits, which for years have appeared satisfactory, suddenly develop large cracks. As an example, our sickness policy seemed excellent, but recently a nearly catastrophic spell hit us when three of us were off with long illnesses. This brought home to us that our arrangements needed exploring—and we hope to arrive at a better answer.

The ratio of staff to case load, the possibility of increasing work load, by reason of adding work or the new opening of operating rooms, etc., the most efficient use of present staff are problems which are always present. Retirement policy has to be worked out although most of our group are not really seriously considering retirement in the next year or so.

The political aspects of the group are for the most part handled by an elected executive of four, plus the director of the hospital department *ex officio*. The executive are elected annually, and select from their own number a chairman, secretary, office supervisor, and financial chairman. Meetings of the executive are called as often as necessary, and a general group meeting is held about once a month, but at least every seventy days.

Members of our group are encouraged to take part in the broader aspects of both medical and non-medical community affairs. One of our group acts as director of the hospital department, is on the hospital medical board, is Clinical Associate Professor at the University, and acts on the British Columbia executive of the Canadian Medical Protective Association. Another has acted for the past several years on the Canadian Medical Association, B.C. Division, Executive, a third is on the Board of Directors of S.A.M.S. The B.C. Division of the Canadian Anaesthetists' Society executive has two or three representatives at all times. Participation in community affairs is encouraged.

The history and experience of a large group of anaesthetists over the past decade may be of interest, and we trust of use to the specialty of anaesthesia, which, perhaps more than any other branch of medicine, tends to be most effectively practised in partnership.

All aspects of the anaesthesiologist's relationship to his patients, surgical colleagues, hospital administrator, the community in general and fellow specialists benefit from this type of association.

BOOK REVIEWS

A SYNOPSIS OF ANAESTHESIA. By J. Alfred Lee. Fourth edition. Bristol: John Wright and Sons Limited [Toronto: The Macmillan Company of Canada]. $4.70.

The fourth edition of Lee's *A Synopsis of Anaesthesia* has arrived at last. The seemingly long wait has been well rewarded for the author has succeeded in maintaining the high standard of the previous editions to which he has dedicated himself.

A very complete revision of the text has been carried out. Many references to recent literature bring the material very up to date, a difficult thing to do when producing a textbook of this size. Two chapters have been added, one on the phenothiazine derivatives and one on induced hypothermia. The author has considerably elaborated some chapters, in particular the chapter on anaesthesia for thoracic surgery, making reference to the new and ingenious tubes and devices used in this branch of anaesthesia. There are many new illustrations and a greater number of them are on coated stock which has made them clearer.

The chapter on "Some Anatomical and Physiological Notes" remains fundamentally as it was, and it seems difficult to improve it without increasing the size of the book. This is unfortunate for it can only really be used as a source of headings from which point the reader should consult the standard texts to complete his knowledge.

One must finally compliment the author on the very complete index to his book which is one of the best that this writer has seen.

E. York

SYMPOSIUM ON PULMONARY VENTILATION. Edited by R. P. Harbord and R. Woolmer. Altrincham, Cheshire: John Sherrat & Son. 12*s*. 6*d*.

This modest volume contains a verbatim report of a meeting, held under the auspices of the *British Journal of Anaesthesia*, at which British authorities on subjects related to pulmonary function presented papers, which were then discussed by the group as a whole.

Varying in standard of material, this symposium is predominately of high calibre, forming interesting reading for those already interested but not being of much value as a teaching primer.

H. Barrie Fairley

MEETINGS

INTERNATIONAL ANESTHESIA RESEARCH SOCIETY, 34th CONGRESS

Shoreham Hotel, Washington, D.C.

April 3–7, 1960

CANADIAN ANAESTHETISTS' SOCIETY, WESTERN DIVISIONS

Empress Hotel, Victoria, B.C.

April 28–30, 1960

SECOND WORLD CONGRESS OF ANAESTHESIOLOGISTS
World Federation of Societies of Anaesthesiologists

Royal York Hotel, Toronto, Ont.

September 4–10, 1960

SECOND WORLD CONGRESS OF ANAESTHESIOLOGISTS
DEUXIÈME CONGRÈS MONDIAL DES ANESTHÉSISTES

FORM C **FORMULE C**

APPLICATION FOR ENROLMENT
DEMANDE D'INSCRIPTION COMME MEMBRE

TO:
A: The Manager,
Second World Congress of Anaesthesiologists,
2 College St.,
Toronto 5, Ontario, Canada.

I wish to enrol as an active member of the Second World Congress of Anaesthesiologists, to be held in Toronto, Ontario, Canada, September 4th to 10th, 1960.

Je désire m'inscrire comme membre actif du Deuxième Congrès Mondial des Anesthésistes, qui aura lieu à Toronto, Ontario, Canada, du 4 au 10 septembre, 1960.

PLEASE PRINT IN BLOCK LETTERS — EN CAPITALES D'IMPRIMERIE, S'IL VOUS PLAIT

SURNAME
NOM .....

CHRISTIAN OR GIVEN NAMES
PRENOMS .....

ADDRESS
ADRESSE .....

.....

.....

ACADEMIC APPOINTMENT OR AFFILIATION
SITUATION ACADÉMIQUE OU ASSOCIATION .....

LANGUAGE PREFERENCE
LANGUE CHOISIE .....

Are you attending as the representative of any Government, Society or Organization?
If so, please specify .....

.....

Participez-vous comme représentant d'un Gouvernement, d'une société ou d'une organisation quelconque? Si oui, veuillez préciser

.....

I shall be accompanied by the following associate members
Je serai accompagné par les membres associés suivants .....

.....

(Names) (Noms)

FEES: Active Membership: $25.00 Canadian payable before Feb. 1, 60, or $30.00 if submitted after Feb. 1, 60.

Associate Membership: $10.00 Canadian payable before Feb. 1, 60, or $15.00 if submitted after Feb. 1, 60.

I enclose money order or bank draft for $..... payable in Canadian funds.

Enrolment fees will be returned to those unable to attend the Congress who so notify the Manager prior to August 1, 1960.

DROITS D'INSCRIPTION: Membre Actif: Dollars canadiens $25.00 à toucher avant le 2 fevrier 60. $30.00 si présenté après le 2 février 60.

Ci-joint un mandat ou un chèque pour $..... payable en dollars canadiens.

Les droits d'inscription seront remboursés à ceux qui ne pourront pas assister au Congrès, et qui en avertiront l'Organisateur avant le 1er août 1960.

Date .....

NOTE: Forms for requesting hotel accommodations will be sent at a later date to all who return this form.

Signature .....

NOTE: Les formes pour les réservations d'hôtel seront envoyées plus tard aux personnes qui auront retourné cette forme.

SEGUNDO CONGRESO MUNDIAL DE ANESTESIOLÓGOS
ZWEITER WELTKONGRESS DER ANAESTHESIOLOGEN

FORMA C **FORMULAR C**

SOLICITUD DE PERMISO PARA PARTICIPAR EN EL CONGRESO
ANMELDUNG ZUR TEILNAHME

DIRIGIDA A:

AN: The Manager,
Second World Congress of Anaesthesiologists,
2 College St.,
Toronto 5, Ontario, Canada.

Deseo registrarme como miembro activo del Segundo Congreso Internacional de Anestesiólogos que tendrá lugar en Toronto, Ontario, Canadá, desde el día 4 hasta el día 10 de septiembre de 1960.

Ich wünsche als aktives Mitglied am zweiten Weltkongress der Anaesthesiologen teilzunehmen, der vom 4. bis 10. September 1960 in Toronto, Ontario, Canada, stattfinden wird.

HÁGASE EL FAVOR DE IMPRIMIR EN MAYÚSCULAS — BITTE IN DRUCKSCHRIFT AUSZUFÜLLEN

APELLIDO
FAMILIENNAME .....

NOMBRE DE BAUTISMO O DE PILA
VORNAME(N) .....

DIRECCIÓN POSTAL
ADRESSE .....

DESIGNACIÓN O AFILIACIÓN ACADÉMICA
AKADEMISCHE STELLUNG ODER BEZIEHUNG .....

IDIOMA PREFERIDO EN EXPRESARSE
BEVORZUGTE SPRACHE .....

¿Piensa usted asistir en capacidad de representante de un gobierno, una sociedad u organización? En caso afirmativo, tenga la bondad de especificarse.
Nehmen Sie als Vertreter einer Regierung, Vereinigung oder Organisation teil? Im Bejahungsfalle bitte um genaue Angabe.

.....

Los siguientes colegas me acompañarán .....

.....

(Nombres)

Ich werde von den folgenden Mitgliedern begleitet werden .....

.....

Namen)

CUOTAS:

Miembro activo: $25.00 (moneda canadiense) pagaderos antes del 1 de febrero de 1960, o $30.00 (moneda canadiense) entregados después del 1 de febrero de 1960.

Miembro asociado: $10.00 (moneda canadiense) pagaderos antes del 1 de febrero de 1960, o $15.00 (moneda canadiense) entragados después del 1 de febrero de 1960.

Adjunto letra de cambio o giro en $..... pagaderos en moneda canadiense.

Los gastos de matrícula seran reembolsados á todos aquellos que se vean incapacitados de atender al Congreso, siempre que asi se lo notifiquen al Director del Congreso antes del primero de agosto de 1960.

Fechada en
Datum .....

NOTA: Más tarde se mandarán formularios para acomodaciones de hotel, a quienes devuelvan esta hoja.

GEBÜHREN: Für aktive Mitglieder— $25.00 (kanad.) zahlbar vor dem 1. Feb. 1960 oder $30.00 (kanad.) überwiesen nach dem 1. Feb. 1960.

Für teilnehmende Mitglieder— $10.00 (kanad.) zahlbar vor dem 1. Feb. 1960 oder $15.00 (kanad.) überwiesen nach dem 1. Feb. 1960.

Beiliegend Postüberweisung oder Bankwechsel für $..... zahlbar in kanadischer Währung.

Anmeldegebühren werden im Falle einer Verhinderung der Teilnahme am Kongress zurückerstattet, wenn die Kongressleitung vor dem 1. August 1960 hiervon benachrichtigt wird.

Firma
Unterschrift .....

ANMERKUNG: Formulare zur Anforderung von Hotelzimmern werden später an alle Einsender dieses Formulars versandt werden.

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pulls the "inoperable" emergency patient out of shock . . . readies patient for surgery . . .

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There is a difference

It's difficult to distinguish between the Monarch butterfly (on the left) and the almost identical Viceroy. Yet the Monarch, because it secretes a fluid which is distasteful to birds, is therefore immune from their attacks — while the Viceroy depends only on its resemblance to the Monarch to keep from being eaten.

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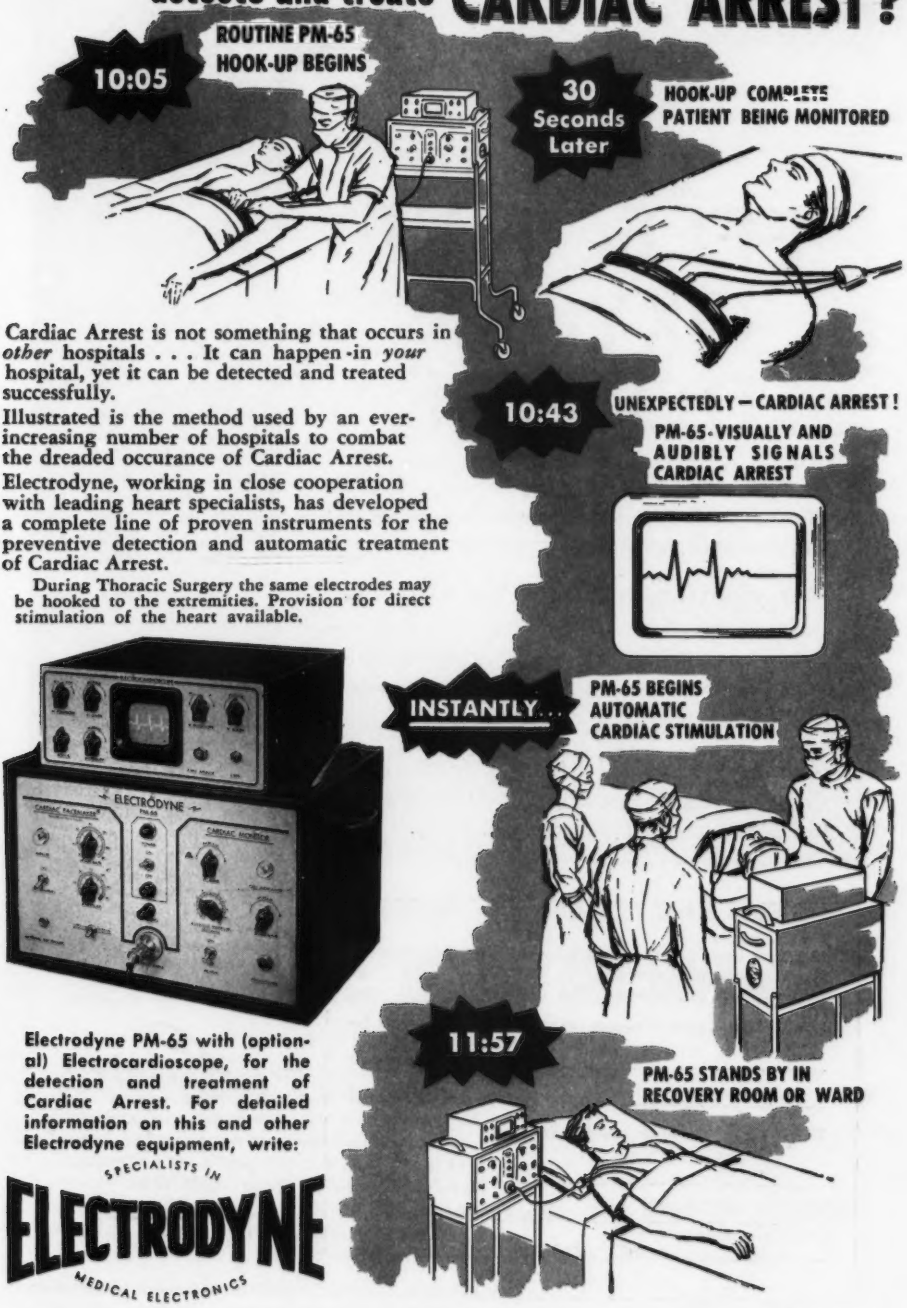

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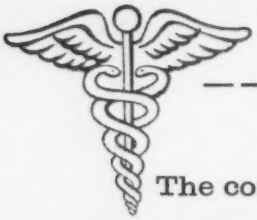

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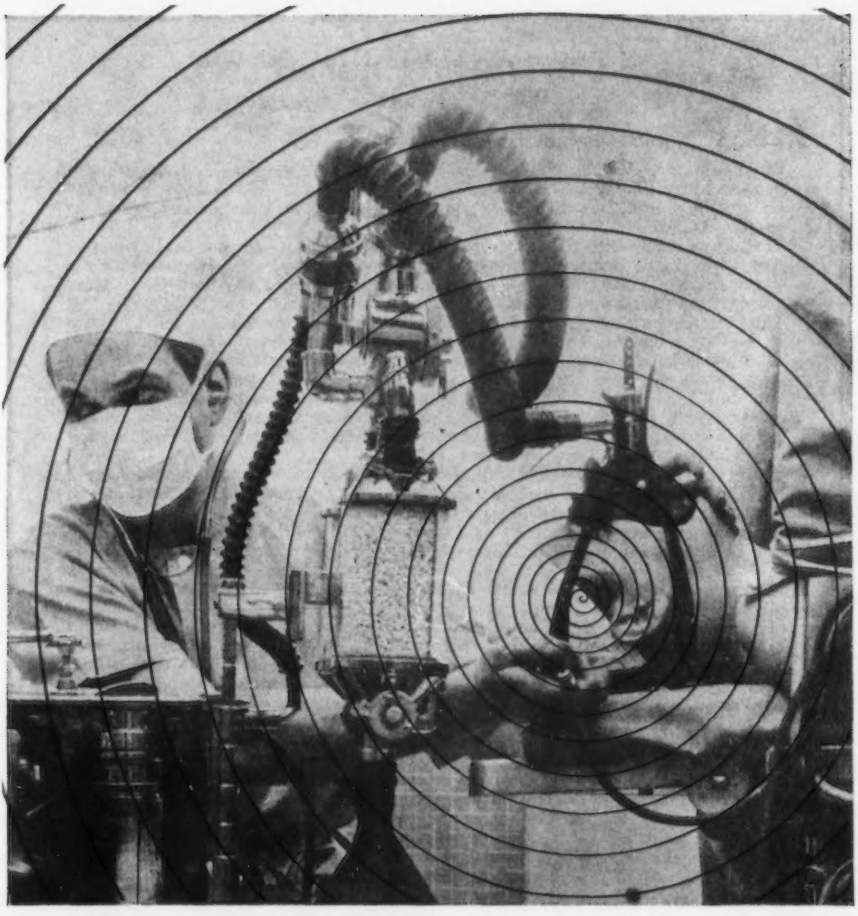

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VINETHENE is a superior induction agent prior to ethyl ether. Its action is rapid and smooth. Excitement or nausea is rarely encountered. Muscular relaxation is good. Such benefits, established for a quarter of a century, also recommend VINETHENE for short operative procedures and as a complement to nitrous oxide or ethylene. VINETHENE is easily administered via open, semi-closed or closed methods.

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ANAESTHETIC NEWS

The History of Anaesthesia

PART 1 *From Adam to Humphrey Davy*

Every minute of the day, in hospitals the world over, surgeons and anaesthetists perform intricate and critical operations. Their patients are old and young, healthy and totally infirm, but their lives are protected and extended immeasurably by modern science.

Through research, the science of anaesthesia has reached a high standard of perfection. It might be said that its origin can be traced even to the beginning of the world and the creation of Eve when, the Bible says: "And the Lord God caused a deep sleep to fall upon Adam, and he slept."

The early centuries are spotted only with the occasional mention of relief of pain in the form of anaesthesia. A Babylonian clay tablet describes the use of herb drugs as an anaesthetic in 2250 B.C. Throughout the Middle Ages many attempts were made to alleviate pain, mainly involving "black magic" and very little logic. It was not until 1513 that the first practical method was attempted. During that year, monks used a method of inhaling alcoholic fumes to ease suffering prior to and during surgical intervention. However, over two centuries elapsed before the first dependable means replaced these early efforts.

Until the experiments of Joseph Priestley there were isolated cases of partial success, but it was the discovery of carbonic acid gas, oxygen and nitrous oxide in 1771 and 1772 that actually led to anaesthesia as we know it today.

Priestley's experiments led to the first apparatus to be designed for the administration of nitrous oxide. The machine was fabricated by Humphrey Davy in 1779. Davy's first experiments with this gas encouraged many others to further discoveries. For instance, William Allen, lecturer on Chemistry at Guy's Hospital, wrote in his diary in March, 1800:

"Present, Astley Cooper, Bradley, Fox and others. We all breathed the gaseous oxide of azote. It took a surprising effect upon me, abolishing completely at first all sensation; then I had the idea of being carried violently upward in a dark cavern with only a few glimmering lights. The company said that my eyes were fixed, face purple, veins in the head very large, apoplectic stertor. They were all much alarmed, but I suffered no pain and in a short time came to myself."

After these early experiments, success was followed fast by further success. Anaesthesia, and the equipment to administer it, were developed through the centuries until they reached the high standards of the British Oxygen Company products of today. But progress does not stop here. Every year new discoveries, through continuing research, lead to further perfection of the B.O.C. Boyle Machine and anaesthetic sundries. Our part in the struggle for health and longevity is continuous in the provision of the finest anaesthetic equipment and medical gases . . . and our most important product . . . DEPENDABLE SERVICE.

THE WRIGHT RESPIROMETER

A form of portable gas meter, the Wright respirometer is suitable for use where smallness and light weight, together with low air resistance and low inertia, are important considerations. It is particularly suitable for respiratory measurements in anaesthesia, in oxygen therapy, and as a research tool of general application in respiratory physiology. The instrument is strongly made and the moving parts are no more delicate than those of a standard watch movement.

For respiratory measurements in anaesthesia . . . always specify the Wright Respirometer.

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CANADIAN ANAESTHETISTS' SOCIETY
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Name _____

Address _____

Education (Universities only—Dates and Degrees) _____

_____

_____

Internships _____

_____

_____

Post-Graduate Training in Anaesthesia (Location and Dates) _____

_____

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Specialist Qualifications in Anaesthesia (state Dates and if by Examination or otherwise) _____

_____

Appointments in Anaesthesia (Past and Present—Full Time or Part Time) _____

_____

Professional Memberships _____

*Are you a member of the Canadian Medical Association or of L'Association des Médecins de la Langue Française de l'Amérique du Nord? _____

Publications (Please attach list if necessary) _____

_____

Signature of Applicant _____

Proposed by _____

Seconded by _____

Fees: Active Members—$25.00 per annum; Members Elect (Residents in Anaesthesia)—$4.00 per annum.

Please make cheques payable to:
"The Canadian Anaesthetists' Society"
and forward to Secretary-Treasurer—178 St. George St., Toronto, Ont.

*Membership in one of these associations is a prerequisite to membership in the Canadian Anaesthetists' Society.

LA SOCIÉTE CANADIENNE DES ANESTHESISTES
DEMANDE D'ADMISSION

Nom _____

Adresse _____

Etudes (Universitaires seulement—Degrés et dates) _____

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Internats _____

_____

_____

Cours post-universitaires en Anesthésie (lieu et date) _____

_____

_____

Qualification de Spécialiste en Anesthésie (Donner les dates, est-ce par examen ou non?) _____

_____

Nominations en Anesthésie (Passées, présentes, temps complet ou partiel) _____

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Affiliations professionnelles _____

*Etes-vous membre de l'Association Médicale Canadienne ou de l'Association des Médecins de Langue Française de l'Amérique du Nord? _____

Publications (S'il vous plaît, inclure une liste si nécessaire) _____

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Signature du candidat _____

Proposé par _____

Secondé par _____

Cotisation: Membres—$25.00 par année; Membres élus (Résidents en Anesthésie)—$4.00 par année.

S.V.P. faire les chèques payable à:

"La Société Canadienne des Anesthésistes"
et envoyer à: Secrétaire-Trésorier, 178 St. George St., Toronto, Ont.

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THE SECOND WORLD CONGRESS OF ANAESTHESIOLOGISTS

TORONTO, CANADA

SEPTEMBER 4 to 10, 1960

GENERAL INFORMATION

The Second World Congress of Anaesthesiologists will be held at the Royal York Hotel, Toronto, Ontario, Canada, from September 4th to 10th, 1960. This quinquennial function of the World Federation of Societies of Anaesthesiologists will be convened under the auspices of the Canadian Anaesthetists' Society, as host. The Congress will be open to all physicians interested in Anaesthesiology.

SCIENTIFIC PROGRAMME

The Scientific programme, divided into plenary and concurrent sessions, will extend from Monday, September 5th, to Friday, September 9th, 1960.

The Programme Committee invites contributions to the scientific programme in the form of reports on current clinical investigation, basic research, teaching of anaesthesia and organization of departments.

The Committee is anxious to receive papers from all countries, but it is necessary to strictly limit presentations for the plenary sessions to twenty minutes including presentation of slides or brief illustrative movies, and to ten minutes for the concurrent sessions.

Discussion of the twenty-minute papers will be limited to two five-minute periods by designated speakers and there will be no discussion of the ten-minute presentations.

Submission of Papers—Those offering papers for consideration should complete an Application Form and return it with an abstract of 250 words to reach this office by December 31st, 1959. The full text of the paper must be submitted in quadruplicate by March 31st, 1960.

Panel Discussions on selected subjects will be arranged.

Cinema Exhibits—There will be a special room for continuous showing of films on anaesthesia. Those offering films for consideration should complete an Application Form which should reach this office not later than December 31st, 1959.

Official languages will be English, French, German and Spanish and simultaneous translation will be provided at all sessions.

Forms may be obtained from

The Manager
WORLD CONGRESS OF ANAESTHESIOLOGISTS
2 College Street
Toronto 2, Ont.

THE SECOND WORLD CONGRESS OF ANAESTHESIOLOGISTS

and Assembly of

The World Federation of Societies of Anaesthesiologists

TORONTO, CANADA

SEPTEMBER 4 to 10, 1960

Communications should be addressed to

The Chairman
Organizing Committee
World Congress of Anaesthesiologists
178 St. George Street
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THE CANADIAN ANAESTHETISTS' SOCIETY JOURNAL

Editorial Policy

The Canadian Anaesthetists' Society Journal is published quarterly by the Canadian Anaesthetists' Society Inc. Original articles are accepted for publication on the understanding that they are contributed exclusively to this journal and become the property of the Canadian Anaesthetists' Society. Articles are subject to such alteration as the Editor in his absolute discretion may deem necessary, but no major alterations will be made without consent of the Author.

Manuscripts

Articles should be typewritten in double space on one side of the paper only. Pages must be serially numbered, and each page should carry at its head the name of the author and the title of the article in full or in an appropriate abbreviation. The article should be concluded by a summary which will be intelligible without reference to the main text. All articles should be accompanied by a résumé presenting the important features in short form, for translation into the French language. French-speaking authors should provide this résumé in the French language.

References to the literature should be clearly indicated in the text by arabic numerals in brackets, thus (4). They should be set out in numerical order at the end of the article, typed in double space, as follows:

4. Griffith, H. R. & Johnston, G. E. The Use of Curare in General Anaesthesia. Anesthesiology 3: 481 (1942).

References to books will state in order: Name of Author, Title of Book, Edition, Place of Publication, Publisher, Year of Publication, such as:

Labat, G. Regional Anesthesia. 1st ed., Philadelphia: Saunders (1922).

The names of all authors will be given in the first instance in each reference. In further references to the same authors the abbreviated form "Griffith *et al.*" may be used.

Illustrations

Photographs should be unmounted glossy prints. Drawings and charts should be in black India ink on white paper. Reproductions in colour will be undertaken only at the expense of the author. All illustrations must be referred to in the text by Arabic Numerals (thus—Figure 3) the corresponding Arabic Numeral being clearly marked on the back of the illustration, together with the name of the author and the title of the article. Legends for illustrations must be typewritten in double space on a separate sheet of paper and clearly marked with the numerals corresponding to the appropriate illustrations.

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